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AGRICULTURE

84–0001. Braun, H. E.; Frank, R.; Hoffman, I. **Estimation of MH in Ontario Tobacco Between 1968-1982 Using a Rapid Screening Procedure.** *Tobacco International* 185(16): 30-31, August 5, 1983.

A rapid screening procedure, based upon the reduction of maleic hydrazide (MH) to hydrazine followed by distillation and reaction with *p*-dimethylaminobenzaldehyde, is described for estimating MH residues in tobacco. The limit of detection approximates field applications of 0.5 kg/ha and the method has been used to test up to 60 samples/day by one technician. This testing program was effective in monitoring the use of MH on Ontario-grown tobacco in accordance with an agreement between Ontario tobacco growers and tobacco processors. Of the 44,000 randomly selected tobacco samples tested between 1968 and 1982, 52 were found to contain detectable levels of MH. (Auth. Abs.)

84–0002. Humphries, B. **Pesticide Residue Continuing Problem; Determines Acceptability of Leaf.** *Tobacco International* 185(15): 132, 134, July 22, 1983.

Because several tobacco-importing countries discriminate against leaf with excessive maleic hydrazide (MH) residues, MH levels are now considered in determining tobacco quality. When the chemical was introduced in the 1950s, it was known that MH persists in cured leaf, but levels were low, averaging 30 to 60 ppm. However, concentrations reached 130 to 140 ppm in the middle to late 1970s. Currently, the majority of samples show residues around 80 ppm or lower, but a relatively small proportion test much higher. For the United States to maintain its quality status in the world market, the MH residue level must be reduced, and farmers are advised to follow the steps recommended by the North Carolina Agricultural Extension Service for effective sucker control at minimal application rates. It is noted that MH is currently the only pesticide-related quality problem, but several new chemicals registered for tobacco, including some for blue mold control, will need careful watching.

84–0003. Ogilvie, I. S.; Kozumplik, V. **Genetic Analysis of Quantitative Characters in Cigar and Pipe Tobacco, *Nicotiana tabacum*. III. Economic Indices: Grade Index, Yield, and Crop Revenue.** *Canadian Journal of Genetics and Cytology* 25(2): 185-189, April 1983.

An investigation of the inheritance of economic indices and chemical analyses of air-cured tobacco varieties indicated that all parameters tested were largely controlled by genes exhibiting additive gene action. A diallel cross of four cigar cultivars and one pipe tobacco (*Nicotiana tabacum* L.) cultivar was analyzed over 3 years for grade index, yield, crop revenue or index, percentage of nicotine, percentage of total alkaloids, and susceptibility to pole rot. In analysis of general combining ability (GCA) and specific combining ability (SCA), GCA was greater than SCA for all variables, although significant SCA effects were also present for all parameters except nicotine and total alkaloids. Reciprocal effects were completely absent. The line Pennbel 69 showed a positive GCA effect for yield, crop index, and infestation by pole rot; and a high negative GCA effect for grade index and percentage of nicotine and total alkaloids. Petit Havane showed a complete reversal of GCA effects for these parameters while the other three cultivars were intermediate. High positive SCA effects for grade index, yield, and crop index, and high negative SCA effects for the percentage of pole rot were shown by crosses of Pennbel 69. (Auth. Abs. Mod.)

84–0004. Tanabe, Y.; Sano, M.; Kawashima, N. **Changes in Free Amino Acids in White and Green Tissues of Variegated Tobacco Leaves During Water Stress.** *Plant and Cell Physiology* 23(7): 1229-1235, October 1982.

Drastic changes in the free amino acid composition occurred in green leaf tissue during a 4-day water stress period, but only very small changes were noted in the white tissue. Variegated tobacco leaves, white on one side of the midrib and green on the other, were detached from the stem and incubated under water stress or turgid conditions to determine any changes in the levels of free amino acids. The most striking difference between the two tissues was the large amount of proline that accumulated in the green tissue but not in the white. An exogenous supply of sucrose increased the proline content in both tissues during water stress. An exogenous supply of glutamic acid increased the proline and asparagine contents in the green tissue, but it increased only the asparagine content in the white tissue during water stress. (Auth. Abs. Mod.)

CHEMISTRY

84–0005. Al-Kubaisi, A. H. **Study on the Pyrogenesis of Polynuclear Aromatic Hydrocarbon in Cigarette Smoke: Pyrolysis of Benzene, Naphthalene and β-Sitosterol.** Master's Thesis, North Carolina Agricultural and Technical State University, Greensboro, 1983, 65 pp.

Data showed that aromatization of the substrate was more important in formation of polynuclear aromatic hydrocarbons (PAHs) in tobacco smoke than polymerization. Samples of benzene, naphthalene, and β-sitosterol were pyrolyzed in a nitrogen atmosphere. The pyrolysates obtained were analyzed for their naphthalene, anthracene, phenanthrene, pyrene, chrysene, and benzo(*a*)pyrene (BaP) contents, i.e., PAHs containing 2, 3, 4 and 5 fused benzene rings. Benzene on pyrolysis gave the following percentages: 0.11 naphthalene, 0.25 phenanthrene, 0.008 anthracene, and traces of BaP. Naphthalene pyrolysis gave 0.032 phenanthrene, 0.002 anthracene, 0.0101 pyrene, 0.0116 chrysene, and 0.0116 BaP; and β-sitosterol pyrolysis gave 3.546 naphthalene, 2.0775 phenanthrene, 0.344 anthracene, 0.49 pyrene, 0.126 chrysene, and 0.141 BaP. Results show that as the number of rings in the substrate increases, the yield of the PAHs in the pyrolysates, especially those containing 4 or 5 rings, increases. The pyrolyses were done under conditions that favored the dimerization and polymerization of the substrate molecule fragments; however, conditions (time and concentration) in cigarette smoke do not favor such reactions. (Auth. Abs. Mod.)

84–0006. Augsten, M.; Depersdorff, J. **Eine Methode zur Bestimmung des Thiozyanats im Serum. [A Method for the Determination of Thiocyanates in Serum.]** *Zeitschrift fur Medizinische Laboratoriums-Diagnostik* 23(4): 226-228, 1982, German.

The thiocyanate content of serum may be determined by allowing it to react with ferric ions to produce ferric thiocyanate. The method permits handling of large batches of samples with sufficient precision. The thiocyanate values thus obtained are suitable for substantiation of smoking. (Auth. Abs. Mod.)

84–0007. Dumas, E.; Vallee, J.-C.; Perdrizet, E. **Etude Comparee du Metabolisme des Acides Amines et Amines Libres Chez Deux Especes de Tabac. [Study Comparing the Metabolism of Amino Acids and Free Amines in Two Species of Tobacco.]** *Physiologie Vegetale* 20(3): 505-514, July-September 1982, French.

Amino acids, and aliphatic and aromatic amines labeled with $^{14}CO_2$, were analyzed in two species of tobacco, *Nicotiana tabacum* c.v. Xanthi and *Nicotiana* RMB$_7$ (which does not flower). In *Nicotiana* RMB$_7$, the deficiency in aromatic amines cannot be explained by a lower synthesis of precursors. In tobacco plants, biosynthesis of aliphatic and aromatic amines is very slow. Aliphatic amines are better labeled than aromatic amines. Putrescine is the first labeled polyamine after 8 hours of $^{14}CO_2$ metabolism. After 36 hours, spermidine and, to a small extent, spermine are labeled. ^{14}C incorporation into amines is not the same in the different parts of the plant. Comparison between the two tobacco species shows that aromatic amines synthesis is strongly disturbed in the nonflowering species *Nicotiana* RMB$_7$.

84–0008. Harriman, E. **Tar Table "Cheats" Are Sued.** *New Scientist* 99(1367): 175, July 21, 1983.

Extensive debate over machine-measured tar delivery of the Barclay cigarette vs. actual tar yield to the smoker prompted the Federal Trade Commission (FTC) to announce on June 25, 1982, that Barclay was no longer in the 1 mg tar category. The FTC, in a suit against British American Tobacco (BAT), claims that the Actron filter imparts seven times more tar to smokers than to testing machines due to smokers' tendency to crush the "aerodynamic" grooves in the filter tip. It is also claimed that smokers' lips block the ends of the grooves with the same effect. Spokespersons for Brown and Williamson, BAT's American subsidiary, claim that cotinine levels in smokers' blood plasma after smoking Barclay, Cambridge, and Carlton matched the published deliveries of nicotine determined through the FTC's testing method. However, FTC-appointed consultants indicate that Brown and Williamson's conclusions were marred by serious methodological shortcomings. See also 84-0012, 84-0336, 84-0339.

84–0009. McCusker, K.; Hiller, F. C.; Wilson, J. D.; Mazumder, M. K.; Bone, R. **Aerodynamic Sizing of Tobacco Smoke Particulate From Commercial Cigarettes.** *Archives of Environmental Health* 38(4): 215-218, July-August 1983.

Aerodynamic sizing of smoke particulates was performed on a variety of commercial cigarettes using a single particle aerodynamic relaxation time (SPART) analyzer. Five cigarettes with filters and five without filters were tested from each brand. Smoke particles from all cigarettes were less than 0.6 μm mass median aerodynamic diameter (the particle number/puff increasing as the cigarette shortened). Filters on commercially available cigarettes reduced smoke particle concentrations by 20 to 96 percent for the cigarettes tested. After filtration of 96 percent of the particles in mainstream cigarette smoke, the concentration of smoke was 10^8 particles/cm^3. Some cigarettes rated as low-tar by the Federal Trade Commission had smoke particle concentrations in the range of those rated as medium-tar. Thus, tar content is not a measure of puff by puff smoke concentration. Also, the ultimate respiratory tract deposition site of smoke particles is not affected by filters, since particle size was not altered by passing smoke through cigarette filters. Findings suggest that estimation of tar delivery to smokers from cigarettes should include puff number information. (Auth. Abs. Mod.)

84–0010. Neri, M. **Determination of the Antioxidant Properties of Tobacco Smoke by Cytochrome C.** *Beitraege zur Tabakforschung International* 12(2): 81-85, June 1983.

The reduction of cytochrome C was used as an index of the antioxidant activity of whole smoke and vapor phase smoke. The method measures the antioxidant activity in smoke by spectrophotometric and potentiometric determinations. Cigarette smoke was placed in direct contact with the buffer solution of the cytochrome C, thus avoiding the collection and extraction necessitated by other systems. The antioxidant activity of smoke was determined by spectrophotometric analysis as a function of the variation in the absorption value of 550 nm caused by the reduced cytochrome, while potentiometric analysis determined the rapid decrease in the reduction potential. Data obtained by the two methods corresponded perfectly with each other and also showed that the above methods are reliable as an index of the antioxidant activity of smoke. Reduction of cytochrome in the smoke of ventilated cigarettes shows a difference in the vapor phase and not in whole smoke. (Auth. Abs.)

84–0011. Norman, V.; Ihrig, A. M.; Larson, T. M.; Moss, B. L. **The Effect of Some Nitrogenous Blend Components on NO/NO$_x$ and HCN Levels in Mainstream and Sidestream Smoke.** *Beitraege zur Tabakforschung International* 12(2): 55-62, June 1983.

Vapor phase nitric oxide (NO) concentrations in mainstream and sidestream smoke correlated well with blend nitrate levels over the range from 0.07 to 3.70 percent. Native and added nitrate fit the same correlation. No measurable amounts of NO_2 were detected in the vapor phase. When the mainstream and sidestream data were extrapolated to zero nitrate level, a large residual amount of NO (approximately 100 μg/cigarette) remained that could be attributed neither to nitrate nor to other nitrogenous blend components of the total volatile base type. Nitrogen-free cellulose cigarettes yielded about 50 μg NO/cigarette, indicating that part of the residual NO probably arises from the oxidation of atmospheric nitrogen. The particulate phase contained small but measurable levels of nitrate that, except for cigarettes with added hydrated Mg (NO_3), did not relate to blend nitrate levels. Mainstream vapor phase HCN level correlated weakly with blend nitrate levels, but other nitrogenous materials, whether native or added, showed no clearly defined precursor-product relationship with smoke HCN yield. The partitioning of HCN between the vapor and particulate phases was not affected by any of the test variables. (Auth. Abs.)

84–0012. Pepples, E.; Froggatt, P. **Cigarette Tar. (Letters).** *New Scientist* 99(1368): 299-300, July 28, 1983.

Controversy over Barclay's actual delivery of tar to the smoker prompted two responses suggesting that the Federal Trade Commission (FTC) reevaluate its testing program; and claiming that previous articles (*New Scientist* 99(1366): 85-87, July 14, 1983, and 99(1367): 175, July 21 1983) unjustly imply cheating. It is argued that FTC testing techniques do not measure tar and nicotine content of the cigarette, but, rather, measure delivery through a smoking machine ventilation system that does not represent the precise yield an individual smoker experiences with any one brand. Barclay's rating as a 1 mg cigarette is defended, as are the work and objectives of the Independent Scientific Committee on Smoking and Health. See also 84-0008, 84–0339.

84–0013. Sakuma, H.; Kusama, M.; Munakata, S.; Ohsumi, T.; Sugawara, S. **The Distribution of Cigarette Smoke Components Between Mainstream and Sidestream Smoke.** *Beitraege zur Tabakforschung International* 12(2): 63-71, June 1983.

Analysis of yields in sidestream and mainstream smoke and sidestream to mainstream distribution ratios (SS/MS) of acidic components, showed (1) major phenolic components were phenol, catechol, and hydroquinone; and (2) major acids were formic, acetic, lactic, glycolic, and succinic. The four cigarette types examined were made from Bright, Burley, Turkish, and domestic (c.v. Matsukawa) tobacco. Compounds that gave SS/MS ratios >1 for all types of cigarettes tested included phenol, cresols, xylenols, guaiacol, formic acid, and acetic acid. Formic and acetic acids were analyzed by gas chromatography following *n*-butyl esterification; acids other than formic and acetic acids and phenols were analyzed after trimethylsilylation. (Auth. Abs. Mod.)

See also, **84–0111**

PHARMACOLOGY AND TOXICOLOGY

84–0014. Abood, L. G.; Grassi, S.; Costanza, M. **Binding of Optically Pure (—)-(^{3}H) Nicotine to Rat Brain Membranes.** *FEBS Letters* 157(1): 147-149, June 27, 1983.

A good correlation was observed between binding (IC_{50}) values and psychotrophic potency of a series of *N'*-alkyl-substituted nicotine analogs. Both a higher affinity and lower affinity site were observed, and stereoselectivity of both sites was similar. The IC_{50} value for (+)-nicotine was threefold greater than that for (—)-nicotine at the higher affinity site and twice that at the lower affinity site, while the psychotropic potency of (—)-nicotine was 20 times that of the (+)-isomer. A 20-fold and 40-fold increase in the IC_{50} values occurred with replacement of methyl by ethyl or isopropyl, respectively; while the psychotropic potency was 1/10 and 1/40 that of (—)-nicotine, respectively. Replacement of methyl by *n*-butyl increased IC_{50} still further and abolished psychotropic activity. Substitution of β-hydroxylethyl in the 6 position of nicotine increased the IC_{50} 50-fold, while decreasing psychotropic activity 30-fold. Although the binding affinity of nornicotine was one-half that of (—)-nicotine, it was only 1/20 as effective as (—)-nicotine in producing prostration. Hexamethonium and α-bungarotoxin (nicotinic cholinergic antagonists) had IC_{50} values around $1X10^{-4}$ M for both the higher and lower affinity sites. Acetylcholine, and the remaining cholinergic agonists and antagonists, also exhibited low binding affinity and did not produce prostration even at 50 times the effective dose of (—)-nicotine.

84–0015. Aceto, M. D.; Awaya, H.; Martin, B. R.; May, E. L. **Antinociceptive Action of Nicotine and Its Methiodide Derivatives in Mice and Rats.** *British Journal of Pharmacology* 79(4): 869-876, August 1983.

Nicotine pyrrolidine methiodine was approximately one-third as active as nicotine in the mouse phenylquinone test, while nicotine pyridine methiodine and nicotine bis methiodine were 100 and 300 times less active, respectively. Three quaternary methiodides of nicotine were prepared and tested for antinociceptive activity in the mouse tail-flick, mouse phenylquinone, and rat tail-flick tests to study the sites of action of nicotine. Following peripheral administration, all three methiodides were inactive in the mouse and rat tail-flick procedures, whereas nicotine was active in both tests, which suggests that nicotine acted centrally. Quaternization of nicotine did not eliminate antinociceptive activity as demonstrated by the intraventricular injection of the methiodides in mice. Nicotine pyrrolidine and bis methiodides were somewhat more potent than nicotine, whereas nicotine pyridine methiodide was considerably less potent than nicotine in the tail-flick procedure. Hexamethonium partially blocked nicotine and nicotine pyrrolidine methiodide, whereas mecamylamine blocked nicotine completely, but nicotine pyrrolidine methiodide only partially. Nicotine may have both central and peripheral actions in the mouse phenylquinone test, whereas nicotine pyrrolidine methiodide may have both nicotine and non-nicotine-like antinociceptive activity. The radiolabeled methiodides were found to penetrate brain poorly (plasma-to-brain ratios >20). The metabolism of the methiodides to nicotine occurred to a greater extent in mice than in rats. (Auth. Abs. Mod.)

84–0016. Adriaenssens, P. I.; Bixler, C. J.; Anderson, M. W. **Isolation and Quantitation of DNA-Bound Benzo(*a*)pyrene Metabolites: Comparison of Hydroxyapatite and Precipitation Procedures.** *Analytical Biochemistry* 123(1): 162-169, June 1982.

A comparison of the specific activity (pmol/mg DNA) of benzo(*a*)pyrene metabolite—DNA adducts in DNA, isolated by both procedures from the lung, liver, and forestomach of A/HeJ mice given oral benzo(*a*)pyrene, showed that total DNA-associated radioactivity/mg DNA was higher with the precipitation procedure than with the hydroxyapatite method. This was partly due to large amounts of early eluting peaks in the high pressure liquid chromatography analyses of digests of DNA isolated by the precipitation procedure. However, the specific activities obtained for binding of benzo(*a*)pyrene-7,8-dihydrodiol-9,10-epoxide isomers to DNA isolated by the two methods were in close agreement. The hydroxyapatite procedure resulted in the isolation of purer DNA and required less time and sample handling than the precipitation method. For isolation of DNA from whole tissue for carcinogen—DNA adduct analysis, these properties make the hydroxyapatite procedure superior to the precipitation method. It is stated that these findings have important implications when comparing the results of DNA-binding studies that employ different DNA isolation procedures. (Auth. Abs. Mod.)

84–0017. Alster, P.; Berlin, T.; Bohman, S.-O.; Nowak, J. **Nicotine Inhibits Prostaglandin Synthesis in Human Kidney Microsomes.** *Acta Physiologica Scandinavica* 117(4): 581-583, April 1983.

Addition of nicotine to renal cortical and renal medullary microsomal incubates resulted in a dose-related decrease in amount of labeled prostaglandins (PG) formed. Relative distribution of the three principal PGs formed was not affected, however, suggesting that nicotine acts at the cyclooxygenase level. Indomethacin also inhibited synthesis of PGs, with a potency about 1,000 times higher than that of nicotine.

84–0018. Bain, B. J. **Some Influences on the ESR and the Fibrinogen Level in Healthy Subjects.** *Clinical and Laboratory Haematology* 5(1): 45-54, 1983.

In men, the rise in fibrinogen with age was minimal in nonsmokers, but showed a steady increase with age in smokers. In women, the difference between smokers and nonsmokers was not consistent and was significant only in the age group 30 to 39 where smokers had higher fibrinogen levels than nonsmokers. In healthy subjects the erythrocyte sedimentation rate (ESR) is higher in women than in men, and in both sexes a rise with age occurs that may be due to elevation of the fibrinogen level. Results of serum analysis of 537 healthy volunteers (311 males, 226 females) showed that the ESR correlated both inversely with the hemoglobin level and directly with the fibrinogen level, thus explaining the difference in ESR between men and women, and the major part of the age-related increase in the ESR. The ESR did not differ in smokers and nonsmokers, nor was it related to alcohol intake. Oral contraceptive intake did not influence either the fibrinogen level or the ESR. The correlation of ESR and fibrinogen concentration in healthy subjects suggests that physiological variation in the ESR is determined by the fibrinogen concentration as well as the hemoglobin level. (Auth. Abs. Mod.)

84–0019. Benowitz, N. L.; Hall, S. M.; Herning, R. I.; Jacob, P., III; Jones, R. T.; Osman, A.-L. **Smokers of Low-Yield Cigarettes Do Not Consume Less Nicotine.** *New England Journal of Medicine* 309(3): 139-142, July 21, 1983.

Contrary to suggestions that low-yield cigarettes reduce tar and nicotine exposure of smokers, it was found that smokers of low-nicotine cigarettes do not consume less nicotine. The actual nicotine content of commercial cigarettes with different nicotine and tar yields was determined with smoking machines, while actual nicotine intake was determined by blood cotinine levels in 272 subjects smoking various brands of cigarettes. Low-yield cigarette tobacco did not contain less nicotine; in fact, the nicotine concentration in tobacco correlated inversely ($r=-0.53$, $p<0.05$) with the concentration measured by machine. Blood cotinine concentrations correlated with the number of cigarettes smoked/day but not with the machine measured nicotine yield. Only 3.8 to 5.0 percent of total variance in blood cotinine was contributed by nicotine yield. (Auth. Abs. Mod.)

84–0020. Blanck, A.; Rane, A.; Toftgard, R.; Gustafsson, J.-A. **Biotransformation of Benzo(*a*)pyrene and 7-Ethoxyresorufin and Heme-Staining Proteins in Microsomes from Human Fetal Liver and Placenta.** *Biochemical Pharmacology* 32(10): 1547-1552, May 15, 1983.

The livers from fetuses of smokers displayed a somewhat higher enzyme activity toward benzo(*a*)pyrene (BaP) than the livers from nonsmokers' fetuses, suggesting that maternal smoking is a prerequisite for the formation of BaP metabolites in the placenta. Fetal liver and placental microsomes were assayed for metabolism of BaP and 7-ethoxyresorufin (7-EOR). The BaP metabolites were separated using high performance liquid chromatography. Following incubations with fetal liver microsomes, in general only phenols were detectable, while after incubations with midgestational as well as term placentas from smoking women, the 9,10-, 4,5-, and 7,8-dihydrodiols were also formed. No quinones were detected. Placental microsomes from nonsmokers did not catalyze the formation of BaP metabolites. The 7-EOR *O*-de-ethylase activity was in the same range in the fetal livers as in the midgestational placentas, but activities were somewhat higher in the placentas from smokers. Microsomal proteins from fetal livers and midgestational and term placentas were stained for heme to detect multiple forms of cytochrome P-450. In fetal liver microsomes, five protein bands were found in the molecular weight region of 46,000 to 60,000. In the placentas, two bands were seen in the 46,000 to 52,000 region. No correlation between enzymatic activities in vitro and intensity of any specific protein band was observed for the fetal livers or placentas studied. (Auth. Abs. Mod.)

84–0021. Brockhaus, A.; Freier, I.; Ewers, U.; Jermann, E.; Dolgner, R. **Levels of Cadmium and Lead in Blood in Relation to Smoking, Sex, Occupation, and Other Factors in an Adult Population of the FRG.** *International Archives of Occupational and Environmental Health* 52(2): 167-175, July 1983.

Smoking accounted for 55 percent of the variance in blood cadmium (CdB) levels, while smoking and sex interactions accounted for 15 percent of the variance in blood lead (PbB) levels among 579 residents (aged 60 to 65 years) in Cologne and two nearby cities. Average CdB levels in cigarette smokers are three to four times higher than in nonsmokers (geometric means: nonsmokers, 0.44 µg/l; ≤ 10 cigarettes/day, 1.16 µg/l; >10 cigarettes/day, 1.85 µg/l). Results indicate that, with regard to the internal dose, cadmium exposure via smoking may contribute even more than does exposure via food. PbB levels (geometric mean, 8.49 µg/100 ml; range, 2.9-30.3 µg/100 ml) are in the acceptable range as defined by CEC reference values. Male smokers have slightly higher average PbB levels than male nonsmokers. In women, average PbB levels are lower than in men. Passive smoking by 414 nonsmokers resulted in no change in PbB or CdB levels. (Auth. Abs. Mod.)

84–0022. Caderni, G.; Dolara, P. **Detection of Mutagens in Human Urine by Means of XAD-2 Adsorption-Desorption Techniques and Bacterial Mutagenesis.** *Pharmacological Research Communications* 15(8): 775-782, September 1983.

Smokers' urine was tested for mutagenic activity with the *Salmonella* microsome assay. Urine was passed through columns of XAD-2 resin and the adsorbed organic compounds were eluted with either methylene chloride or acetone, and resuspended in DMSO after evaporation of the solvents. Volumes up to 50 ml of urine/plate were tested with and without metabolic activation in vitro. Linear dose—response curves were observed with both solvents, and acetone was (in most cases) more efficient than methylene chloride in removing mutagenic compounds from the columns and equally efficient in some others. The urine concentrates exhibited variable toxicity on *Salmonella* cells, the number of survivors being as low as 10 percent, but methylene chloride did not show a notable advantage over acetone in terms of cellular toxicity. To increase the sensitivity of the method, acetone as solvent and high volumes of urine/plate are recommended. (Auth. Abs.)

84–0023. Casanueva, F. F.; Betti, R.; Cella, S. G.; Mueller, E. E.; Mantegazza, P. **Effect of Agonists and Antagonists of Cholinergic Neurotransmission on Growth Hormone Release in the Dog.** *Acta Endocrinologica* 103(1): 15-20, May 1983.

Activation of cholinergic nicotinic receptors by nicotine (6 mg) failed to alter canine growth hormone (cGH) concentrations, and pretreatment with the nicotinic receptor blocker mecamylamine (5 mg 30 minutes before) did not counteract the GH-releasing effect of eserine. Administration of the cholinesterase inhibitor eserine (0.5 mg), or the cholinergic muscarinic receptor agonist oxotremorine (0.25 mg), in unanesthetized dogs, induced a clear-cut rise in plasma cGH levels. Complete suppression of the GH-releasing effect of eserine and oxotremorine was induced by blockade of cholinergic muscarinic receptors by atropine but not by scopolamine-N-butyl bromide, an anticholinergic drug that does not cross the blood brain barrier. Other cholinomimetic drugs (pilocarpine, 4-aminopyridine, carbachol, and bethanechol) failed to induce a rise in plasma cGH concentrations. Results indicate that (1) cholinergic muscarinic but not nicotinic receptors located in the central nervous system (CNS) inside the blood brain barrier play a facilitatory role in tonic cGH release; and (2) pharmacologically distinct muscarinic receptors may exist in the CNS. (Auth. Abs. Mod.)

84–0024. Conibear, S. A. **Long Term Health Effects of Thorium Compounds on Exposed Workers: The Complete Blood Count.** *Health Physics* 44(Supplement 1): 231-237, 1983.

Multiple regression analysis associated current cigarette smoking habit (including ex-smokers who had quit within 12 months) with an increase in almost every complete blood count parameter (but not red blood cell count) among 273 males exposed to thorium between 1940 and 1973 at a monazite sand refining plant. The men were studied at Argonne National Laboratory from 1976 to 1980. In vivo measurements of body burden were made by counting γ-rays emitted by daughter products of retained thorium and by measuring exhaled thoron. Health status was ascertained through questionnaire, physical examination, and clinical laboratory tests. Measured body burden was found to be higher in those with a history of longer exposure. All parameters of the complete blood count were examined for evidence of an effect due to thorium. Comparisons of high and low body burden groups showed that only age and cigarette smoking had an effect on complete blood count parameters. (Auth. Abs. Mod.)

84–0025. Domino, E. F. **Neuropsychopharmacology of Nicotine and Tobacco Smoking.** *Psychopharmacology Bulletin* 19(3): 398-401, 1983.

Nicotine has stimulant and depressant properties at several levels, involving the central and peripheral nervous system, cardiovascular system, and the endocrine system. These actions are further intermingled with personality and other variables to reinforce the use of tobacco. Nicotine's resemblance to acetylcholine and its similar pharmacology are discussed. The effects of nicotine infusion on fast-wave (REM) sleep and transient electroencephalographic activity in cats are explored. Nicotine's rapid tachyphylaxis and interactions with argenine vasopressin and nicotinic cholinergic antagonists are reviewed, as are skeletal muscle tone and endocrine system effects.

84–0026. Eichelbaum, M.; Bertilsson, L.; Sawe, J. **Antipyrine Metabolism in Relation to Polymorphic Oxidations of Sparteine and Debrisoquine.** *British Journal of Clinical Pharmacology* 15(3): 317-321, March 1983.

Data confirm the influence of smoking in antipyrine oxidative biotransformations. Thirty-five healthy subjects (11 smokers and 24 nonsmokers, aged 20 to 48 years), who had been classified as extensive (n=28) or poor (n=7) metabolizers of both sparteine and debrisoquine, were given a single oral dose of antipyrine. Saliva concentrations of antipyrine, and urinary excretion of its three major oxidation metabolites, were measured. All the parameters of antipyrine metabolism that were estimated had similar distributions in both extensive and poor metabolizers. The clearance of antipyrine by the formation of 4-hydroxy-antipyrine and 3-hydroxy-antipyrine, respectively, were closely correlated ($r=0.83$, $p<0.001$), and both were significantly higher in smokers than in nonsmokers. Demethylation of antipyrine also seemed to be influenced by smoking, but not to a statistically significant extent. (Auth. Abs. Mod.)

84–0027. Fox, B.; Bull, T. B.; Oliver, T. N. **The Distribution and Assessment of Electron-Microscopic Abnormalities of Human Cilia.** *European Journal of Respiratory Diseases* 64(Supplement 127): 11-18, 1983.

There was no significant difference in incidence of abnormalities between nonsmokers and ex-smokers in an investigation of the effects of different fixatives on the appearances of cilia, but there was a highly significant increase between these two groups and present smokers ($p<0.001$). The difficulties of assessing the true incidence of electromicroscopic abnormalities of microtubular structure of cilia was overcome by examining large numbers of cilia from each case. Bronchial biopsies were examined from 35 subjects being investigated for various lung diseases, and nasal biopsies from 12 subjects (7 with retinitis pigmentosa (RP), and 5 healthy controls). Numerous pieces of normal-looking bronchial wall from a lobectomy specimen were used to examine the effect of six different fixatives. Results showed 2.9 percent of bronchial cilia (mean of 890 cilia examined) and 2.4 percent of nasal cilia (mean of 808 cilia examined) with microtubular abnormalities. Examining large numbers of cilia established that increased microtubular abnormalities were associated with smoking, chronic pulmonary infection, and carcinoma of the lung. There was a significant increase ($p < 0.001$) in microtubular abnormalities in nasal cilia in RP. The appearances of cilia varied with different fixatives, and the numbers of dynein arms seen and the ease of recognizing radial spokes and microtubules was particularly affected by fixation. (Auth. Abs. Mod.)

84–0028. Fox, R. W.; Samaan, S.; Bukantz, S. C.; Lockey, R. F. **Theophylline Kinetics in a Geriatric Group.** *Clinical Pharmacology and Therapeutics* 34(1): 60-67, July 1983.

The influence of age, sex, and smoking on theophylline disposition was studied in 38 healthy subjects (26 to 81 years of age). There were 8 under age 60 and 30 over 60 years of age, including 28 men (8 smokers) and 10 women (3 smokers). A crossover experimental design was used. A single dose of theophylline elixir (5 mg/kg lean body weight (LBW)) was given as a reference product to all subjects. One week later a sustained-release (SR) theophylline tablet (8 and 6 mg/kg LBW) was administered and serum theophylline concentrations determined by high performance liquid chromatography. Results showed that theophylline elimination was shorter (6.93 and 8.14 hr), total body theophylline clearance was greater (44.39 and 32.97 ml/kg/hr), and the apparent volume of distribution was also greater in the geriatric (over age 60) group (26.29 and 22.97 l). Sex and smoking did not influence any of the parameters studied. In 93 percent of the geriatric subjects, serum theophylline levels of 8 to 20 μg/ml were reached at steady state with the SR tablet. Theophylline dose reduction based on an arbitrary age limit is not, therefore, invariably indicated in the treatment of reversible airway disease. (Auth. Abs.)

84–0029. Furman, B. L.; Twaij, H.A.A. **Evaluation of Combined Adrenal Demedullation and Chronic Guanethidine Treatment as a Sympathectomy Procedure for Metabolic Studies.** *Archives Internationales de Pharmacodynamie et de Therapie* 262(1): 164-176, March 1983.

Combined adrenal demedullation and guanethidine treatment in the rat markedly reduced or abolished pressor and positive chronotropic responses to tyramine, nicotine, or electrical stimulation of the sympathetic spinal outflow; and abolished hyperglycemic responses to nicotine or spinal sympathetic stimulation. Nicotine infusion in intact animals reduced glucose-induced hyperinsulinemia while nicotine infusion in sympathectomized animals produced a marked elevation in glucose-induced hyperinsulinemia relative to that seen in intact animals. The early hyperinsulinemic response to glucose infusion during sympathetic stimulation was markedly greater in sympathectomized rats compared with that seen in intact rats. The sympathectomy procedure did not modify the glycemic responses to adrenaline or insulin or glucose-induced hyperinsulinemia. (Auth. Abs. Mod.)

84–0030. Gardner, M. J.; Tornatore, K. M.; Jusko, W. J.; Kanarkowski, R. **Effects of Tobacco Smoking and Oral Contraceptive Use on Theophylline Disposition.** *British Journal of Clinical Pharmacology* 16(3): 271-280, September 1983.

Assessment of independent and interactive effects of chronic (>6 months) oral contraceptive (OC) use and cigarette smoking on single dose (4-mg/kg) theophylline disposition in 49 healthy women (aged 19 to 30 years, 27 smokers and 22 nonsmokers) showed significant elevations (40 percent) in theophylline plasma clearance in women who smoked. OC use resulted in decreases in clearance of a similar magnitude (28 percent). These factors do not appear to interact with respect to theophylline disposition. The combination of main effects tended to cancel one another (clearance of 49.1 ml/hour/kg ideal body weight for OC nonusers—nonsmokers, vs. 49.7 ml/hour/kg for OC user—smoker). Single-dose exposure to OCs in nonusers did not significantly alter theophylline pharmacokinetics for the group as a whole. However, in the subgroup of smoking subjects, significant decreases in clearance were evident ($p < 0.05$). Analogous results were found for half-life. Volume of distribution was slightly diminished in smokers, but was unaffected in OC users. Areas under the serum concentration-time (AUC) profiles of norgestrel and ethinyloestradiol were examined in 27 women as indices of OC exposure. The smallest values of theophylline clearance were found in subjects with largest AUC of both OC steroids. Appropriate statistical analyses of data that are influenced by multiple factors are discussed, noting that special concern is needed when the factor partitioning process yields subgroups of unequal size. (Auth. Abs. Mod)

84–0031. Gordon, R. E.; Case, B. W.; Kleinerman, J. **Acute NO_2 Effects on Penetration and Transport of Horseradish Peroxidase in Hamster Respiratory Epithelium.** *American Review of Respiratory Disease* 128(3): 528-533, September 1983.

Results show that the tight junctions (TJ) of hamster bronchiolar epithelium are most sensitive to injury by nitrogen dioxide (NO_2), a component of cigarette smoke. Furthermore, NO_2 injury increases endocytic activity of alveolar type I cells, increasing vulnerability to most air pollutants, especially those in cigarette smoke and automobile exhausts. Male golden Syrian hamsters, exposed to 28 ppm NO_2 for 48 hours, received intratracheal instillations of 25 mg horseradish peroxidase (HRP) in 0.2 ml saline following exposure. After 10- to 30-minute incubation periods, lungs were perfusion-fixed and excised, and bronchi, bronchioles, and alveolar tissues were isolated. A second group of NO_2-exposed hamsters was allowed a 48-hour recovery period prior to HRP instillations, and a third group served as air controls. Analysis of airways showed penetration by tracer material of 75 percent of bronchiolar TJ examined after 48-hour exposures. Only 15 percent of bronchial TJ were penetrated. No bronchial TJ were compromised after the recovery period, but 20 percent of bronchiolar TJ remained permeable. The TJ from bronchi and bronchioles of control animals showed no penetration of HRP; HRP penetration of interalveolar TJ was not seen in control animals or in "recovery" animals, but it was observed in 30 percent of the TJ after 48-hour exposures. All penetrated junctions were between adjacent type I pneumocytes. Many pinocytotic vesicles in alveolar type I pneumocyctes containing HRP could be seen 10 and 30 minutes after instillation in animals exposed to NO_2. Control animals showed a similar pattern; however, fewer vesicles were present. Analysis of pinocytotic vesicles for each exposure and control group indicated a 500 percent increase in vesicle number after 48 hours of NO_2 exposure. After 48 hours of recovery, the number of vesicles was 300 percent greater than in control animals. These data were not altered by peroxidase stain. Vesicle size was found to be the same (80 nm in diameter) in NO_2-exposed and control specimens. (Auth. Abs. Mod.)

84–0032. Hansen, J. C.; Kromann, N.; Wulf, H. C.; Alboge, K. **Human Exposure to Heavy Metals in East Greenland. II Lead.** *Science of the Total Environment* 26(3): 245-254, February 1983.

No influence of smoking on blood lead (Pb) levels was found in 175 blood samples from adults from Angmagssalik, East Greenland, or in 130 samples from Aarhus, Denmark. Males from both areas had significantly higher (5 percent) blood lead than females (Eskimoan males 14.8 µg Pb/100 ml, females 12.8 µg Pb/100 ml; Danish males 10.5 µg Pb/100 ml, females 7.7 µg Pb/100 ml). For Danes living temporarily in Greenland, values were 10.5 µg Pb/100 ml in males, and 10.2 in females. Eskimos of both sexes were found to have higher blood lead values than Danes living in the same area. Danish males from Greenland and Denmark were not found to be different, whereas Danish women living in Greenland had a significantly higher (5 percent) mean value than women living in Denmark. In the Eskimo group, but not in the Danish, a weak, positive, but significant age correlation was found. The influence of eating habits (local or imported food) was tested, but not found to influence the blood lead concentrations. (Auth. Abs. Mod.)

84–0033. Hartman, T. G.; Rosen, J. D. **Inhibition of Metabolic Cooperation by Cigarette Smoke Condensate and Its Fractions in V-79 Chinese Hamster Lung Fibroblasts.** *Proceedings of the National Academy of Sciences of the United States of America* 80(17): 5305-5309, September 1983.

Results suggest that the intercellular metabolic cooperation assay, which produced data comparable with that obtained on mouse skin, may be useful for the detection of tumor promoters in complex mixtures such as cigarette smoke condensate, industrial oils, or food. One hundred 2R1 cigarettes were smoked according to Federal Trade Commission guidelines, and the resulting condensate was separated into a water/methanol-soluble fraction (which was further partitioned into acidic and basic components) and an organic solvent-soluble fraction (chromatographed on silicic acid with petroleum ether, benzene/petroleum ether, benzene, ether, and methanol). The following fractions were positive in the metabolic cooperation assay (in decreasing order of activity): organic solvent-soluble, acidic, whole condensate, water/methanol-soluble fractions, and the ether, benzene, and benzene/petroleum ether eluates. The basic fraction and the petroleum ether and methanol eluates were negative. (Auth. Abs. Mod.)

84–0034. Heinonen, T.; Kytoniemi, V.; Sorsa, M.; Vainio, H. **Urinary Excretion of Thioethers Among Low-Tar and Medium-Tar Cigarette Smokers.** *International Archives of Occupational and Environmental Health* 52(1): 11-16, June 1983.

Urinary excretion of thioethers was higher among 26 smokers (13 low-tar, 13 medium-tar) than among 11 nonsmokers ($p=0.002$ with Mann-Whitney U-test). Mean urinary creatinine values (±SEM) were 4.3 ± 0.4 mmol/mol for smokers and 2.8 ± 0.2 for nonsmokers, while individual variation was higher among smokers (from 2.0 to 8.5 mmol/mol creatinine), compared with nonsmokers (from 1.5 to 4.5 mmol/mol creatinine). Comparison between smokers of low-tar (5.4 mg) and medium-tar (16.3 mg) cigarettes showed no differences in urinary thioether levels, either before or after periods of smoking. Individual variation of the medium-tar smokers did not differ markedly from that of low-tar smokers. No clear correlation could be detected between the quantity of cigarettes smoked (10 to 25/day) and the levels of urinary thioethers.

84–0035. Henningfield, J. E.; Miyasato, K.; Johnson, R. E.; Jasinski, D. R. **Rapid Physiologic Effects of Nicotine in Humans and Selective Blockade of Behavioral Effects by Mecamylamine.** In: Harris, L. S. (Editor). *Problems of Drug Dependence, 1982.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse Research Monograph Series No. 43, April 1983, pp. 259-265.

The complexity of responses to nicotine was demonstrated in four male volunteers (all cigarette smokers) by a new microcomputer-controlled behavioral-pharmacologic testing system that recorded changes in pupil diameter in response to nicotine. Subjects received a placebo plus three nicotine doses (0.75, 1.5, and 3.0 mg), and four physiologic measurements were made: (1) skin temperature, (2) electromyogram (EMG) from lower leg, (3) EMG from forearm, and (4) pupil diameter. Nicotine produced dose-related changes in (1) pupil diameter, which showed a bi-phasic increase in response to injections; and (2) skin temperature, which decreased, reaching a maximum about 5 minutes after injection. EMG responses in lower leg were inconsistent; responses in upper arm were consistently altered only by the 3.0 mg dose. Tension in facial and neck muscles visibly increased at high nicotine doses, while the most reliable subjective measure was self-reported drug dose strength that was directly increased by nicotine doses. In a second experiment, mecamylamine (2.5, 5.0, and 10.0 mg) was added to the dosing schedule, producing a dose-related attenuation of the physiological responses to nicotine. It is suggested that mecamylamine may provide a useful adjunct for the treatment of tobacco dependence.

84–0036. Hilgenfeldt, J.; Fratermann, A.; Kijanski, H. D. **An Indicator to Unmask Smokers. A Quantitative Comparison of Thiocyanat, Nicotine and Cotinine in Plasma and Urine to Determine Smoking and Non-Smoking Behaviour in a Sample of Male and Female Adults.** *International Journal of Health Education* 2(2): 46-51, July 1983.

While it was not possible to demonstrate a clear distinction between nonsmokers and light smokers, results show that determination of either thiocyanate in blood serum or cotinine in serum or urine is a valuable objective measure of smoking behavior in persons smoking more than 10 cigarettes/day. Mean and standard deviation of nicotine, cotinine, and thiocyanate measurements in blood plasma and urine 1 and 19 hours after smoking were completed for three groups: nonsmokers (4 men, 11 women); light smokers (4 men, 11 women smoking 3 cigarettes within 5 hours); and heavy smokers (5 men, 10 women smoking 6 cigarettes within 5 hours, or about 20 cigarettes/day). Plasma sample analysis showed (1) all indicators were present in all groups of subjects, including nonsmokers; (2) a considerable overlap between nonsmokers and light smokers; and (3) a sizable difference between heavy smokers and nonsmokers. A critical value of 80 μmol/l thiocyanate in plasma reliably discriminated between heavy smokers and nonsmokers. Results for cotinine in urine showed an overlap between nonsmokers and light smokers, but a clear difference between nonsmokers and heavy smokers. A critical value of 25 μmol/l for cotinine in plasma discriminated between heavy smokers and nonsmokers, while a critical value of 2.5 μmol/l for cotinine in urine was an equally accurate cutoff point. Values of nicotine in urine showed only small differences between the three groups. Further research to determine whether different cutoff points for males and females could improve the distinction between smokers and nonsmokers is suggested.

84–0037. Hill, P.; Haley, N. J.; Wynder, E. L. **Cigarette Smoking: Carboxyhemoglobin, Plasma Nicotine, Cotinine and Thiocyanate Vs Self-Reported Smoking Data and Cardiovascular Disease.** *Journal of Chronic Diseases* 36(6): 439-449, 1983.

Results showed a plateau of plasma nicotine and cotinine concentration at levels above 20 cigarettes/day, with progressive increase of carboxyhemoglobin and thiocyanate corresponding with increasing consumption. Absorption of tobacco smoke constituents was determined by comparing self-reported cigarette consumption and nicotine availability with carboxyhemoglobin and plasma thiocyanate, nicotine, and cotinine levels in 450 smokers. Smokers of low-yield brands (< 1 mg nicotine/cigarette) had lower plasma levels of nicotine and cotinine, but comparable levels of carboxyhemoglobin and thiocyanate. Plasma nicotine bore no relationship to smoke inhalation, while the number of cigarettes consumed/day showed a weak correlation to smoke inhalation. Despite lower nicotine yields, modified behavior enabled smokers to derive similar amounts of carbon monoxide and thiocyanate from both low-yield and high-yield cigarettes, thus counteracting the advantage of low-nicotine brands. The relationship of these parameters to the risk of coronary heart disease is discussed. (Auth. Abs. Mod.)

84–0038. Hoefel, O. S. **Smoking: An Important Factor in Vitamin C Deficiency.** *International Journal for Vitamin and Nutrition Research* Supplement 24: 121-124, 1983.

Average vitamin C plasma levels are 30 to 40 percent lower in smokers compared with nonsmokers, depending on daily consumption of cigarettes. A lower excretion of L-ascorbic acid and higher metabolic turnover have also been reported. Greater differences are evident when the smokers are heavy consumers of alcohol. Maternal levels of vitamin C in breast milk are also lower in smokers. The effects of nicotine, carbon monoxide, toxic aldehydes, cadmium, aromatic hydrocarbons, and other components of cigarette smoke on vitamin C metabolism are briefly described.

84–0039. Ishiyama, I.; Nagai, T.; Toshida, S. **Detection of Basic Drugs (Methamphetamine, Antidepressants, and Nicotine) From Human Hair.** *Journal of Forensic Sciences* 28(2): 380-385, April 1983.

While smokers' hair may contain greater amounts of nicotine, no clear cutoff level was obtained through hair analysis, which showed that nicotine content for smokers and nonsmokers ranged from 18 to 177.2 μg/g. Routine toxicological methods detected methamphetamine, amitriptyline, imipramine, nicotine, and their metabolites in human hair. Animal experiments show that these drugs are found only in sections of hair grown after drug administration; a negative stage indicates that the drug-containing section of hair has not yet come out of the hair follicle. Toxicological hair analysis may help to identify the chronology of intoxication. (Auth. Abs. Mod.)

84–0040. Kanzler, M.; Jaffe, J. H.; Nee, J. **Low Nicotine Cigarettes: Cigarette Consumption and Breath Carbon Monoxide After One Year.** *Clinical Pharmacology and Therapeutics* 34(3): 408-415, September 1983.

In spite of substantial changes in tar and nicotine exposure, carbon monoxide (CO) in breath samples showed remarkably little change during a year-long followup of 96 heavy smokers who had participated in a 12-week brand-switching study. Smokers who, at 12 weeks, had switched to a brand delivering less than half the nicotine of their baseline brand, were offered continued monetary incentives to participate for an additional 6 months, during which time subjects continued to smoke low-nicotine cigarettes (maintenance control subjects increased their tar and nicotine exposure significantly). In the followup study of those who had not changed by more than 50 percent, the original control group, nonswitchers, and moderate switchers did not significantly change nicotine exposure from what it had been at the end of the initial 12-week study. To the extent that CO is involved in smoking-related disorders, switchers derived little if any benefit from switching to low-nicotine brands. However, short-term studies have shown that smokers who switch average lower plasma levels of nicotine, and thus may still derive some benefit from switching brands despite the findings on CO levels. (Auth. Abs. Mod.)

84–0041. Karki, N. T.; Karvonen, J.; Pelkonen, O. **Aryl Hydrocarbon Hydroxylase Activity in Cultured Lymphocytes of Psoriatic Patients.** *Archives of Dermatological Research* 273: 103-109, 1982.

Cigarette smokers in both the psoriatic patient group (n = 68, 32 smokers and 36 nonsmokers) and the control group (n = 39, 17 smokers and 22 nonsmokers) had significantly higher induced and noninduced aryl hydrocarbon hydroxylase (AHH) activities in cultured mitogen-stimulated lymphocytes, compared with nonsmokers. While the induction ratio was also somewhat higher among smokers in the patient group, the difference was not statistically significant. The mean induction ratio in the smoking control population, 8.0, did not differ significantly from that of nonsmokers. There was no significant difference in the mean basal and induced AHH activities between the psoriatics and the controls, although the former showed a slight tendency toward a higher inducibility ratio. In both, the basal and induced AHH values decreased significantly with increasing age. Antipsoriatic treatment (Anthralin, PUVA) did not seem to influence the lymphocyte AHH activities. No association was demonstrated between AHH activities and duration of the disease. (Auth. Abs. Mod.)

84–0042. Kawalek, J. C.; Hallmark, R. K.; Andrews, A. W. **Effect of Lithocholic Acid on the Mutagenicity of Some Substituted Aromatic Amines.** *Journal of the National Cancer Institute* 71(2): 293-298, August 1983.

Data show that lithocholic acid (LA) can alter the metabolism of benzo(*a*)pyrene (BaP) to phenolic metabolites. Alteration was catalyzed by liver 9,000 X *g*-supernatant (S9) fractions from rats treated with either phenobarbitol (PB) (reaction inhibited) or Aroclor 1254 (AC) (reaction enhanced). The effects of LA on the mutagenicity of 24 aromatic amines in the Ames assay were examined. Mutagenicity effects varied with the substrate and the type of supernatant fraction used. With preparations from PB-treated rats, LA caused inhibition of mutagenic response with 16 of 24 compounds tested. With supernatant fractions from AC-treated rats, mutagenicity was inhibited in 10 test compounds, unaffected in 8, and enhanced in 6 others when LA was included in Ames assay mixtures. While no predictions can be made regarding LA effects on Ames assay compounds, results indicate that LA can cause no effect, an inhibitory effect, or enhancement of mutagenicity. Conclusions suggest that environmental or lifestyle factors (i.e., diet, smoking, drugs), which alter the level of microsomal mixed-function oxidases and their natural substrates, play a role in carcinogenesis. Tabulations for LA effect on mutagenicity of monocyclic aromatic amines, aminobiphenyl and related compounds, and polycyclic aromatic amines are provided.

84–0043. Keast, D.; Taylor, K. **The Effects of Chronic Tobacco Smoke Exposure From High-Tar Cigarettes on the Phagocytic and Killing Capacity of Polymorphonuclear Cells of Mice.** *Environmental Research* 31(1): 66-75, June 1983.

Results suggest that fc-receptor site activity of polymorphonuclear (PMN) cells is not significantly affected by tobacco smoke (TS) exposure, but that complement interactions within the phagocytic process are significantly suppressed. Whether this is due directly to complement receptors or to metabolic associations of phagocytosis of the complement-opsoninantigen complex is unclear. Mice were exposed to smoke from high-tar (16 mg tar/cigarette) filtered cigarettes for up to 32 weeks. Intraperitoneal caseinate induction of PMN cells in both TS-exposed and age-matched control mice provided cells for in vitro assessment of both phagocytic and degradative properties. Radioactive *Staphylococcus aureus* cells were opsonized with either immune serum or heat-inactivated (complement deficient) immune serum. Two strains of mice were used in all trials. The PMN cells from BALB/c strain mice were less affected by TS exposure than those of C57 Black mice in both phagocytic and degradative capacities when the *S. aureus* were opsonized with immune serum. However, heat inactivation of complement within the serum reduced the differences between TS-exposed animals and age-matched controls in both strains of mice. The treatment effects of TS exposure were seen at all times tested (from 3 days to 32 weeks). (Auth. Abs. Mod.)

84–0044. Kurup, V. P.; Resnick, A.; Kagen, S. L.; Cohen, S. H.; Fink, J. N. **Allergenic Fungi and Actinomycetes in Smoking Materials and Their Health Implications.** *Mycopathologia* 82(1): 61-64, April 22, 1983.

Cultures of 12 different brands of tobacco cigarettes (both filtered and nonfiltered) yielded 1.5 X 10^4 to 2 X 10^5 colonies of thermophilic actinomycetes/g, predominantly *Thermoactinomyces candidus.* No fungi were isolated from the tobacco products and none yielded any organism when smoke was sampled through the Andersen sampler. Serology for 24 marihuana smokers, 19 cigarette smokers, and 14 controls (no smoking during the past 5 years) showed the presence of antibodies against the thermophilic actinomycetes *T. candidus* (6 tobacco smokers, 13 marihuana smokers, 5 controls) and *T. vulgaris* (nine tobacco smokers, nine marihuana smokers, six controls), while the *M. faeni* antibody was seen in marihuana smokers only (n = 7). The cigarette smokers had no respiratory complaints. *Aspergillus fumigatus* spores were isolated from marihuana smoke, and precipitin antibodies against *Aspergillus* were found in nine marihuana smokers, three cigarette smokers, and two controls. It is suggested that cigarette smokers may inhale more thermophilic actinomycetes spores than nonsmokers, and that studies of their immunological response are essential in determining the relationship between smoking and hypersensitivity pneumonitis.

84–0045. Lambert, C.; Larochelle, P.; Du Souich, P. **Effects of Phenobarbital and Tobacco Smoking on Furosemide Kinetics and Dynamics in Normal Subjects.** *Clinical Pharmacology and Therapeutics* 34(2): 170-175, August 1983.

Tobacco smoking in normal subjects affects the diuretic response to furosemide (F) without modifying kinetics. Five nonsmokers (NS) and five smokers (S) received a single 40 mg intravenous injection of F, and serum and urine were collected. In a second study, the NS group received 100 mg phenobarbital orally for 15 days and then a second dose of F. Cumulative 8-hour urinary excretion of sodium was identical for NS, NS with phenobarbital, and S at 345±30, 357±29, and 353±25 mmol, respectively. Diuresis was 800 ml (20 percent) less in S than in NS. While endogenous creatinine clearance increased from 117±13 to 196±17 ml/min in NS and from 110±12 to 222±30 ml/min in NS with phenobarbital, in the S group clearance increased only slightly (136±23 to 180±34 ml/min). The increase in free water clearance caused by F was smaller in the S group than in the NS group ($p<0.05$). Protein binding and distribution of F were not affected by phenobarbital or tobacco smoking. F clearance was slightly higher in S than in NS, primarily due to a slight increase in extrarenal F clearance. In the NS group, F clearance remained constant after phenobarbital. (Auth. Abs. Mod.)

84–0046. Lindstedt, G.; Eden, S.; Jagenburg, R.; Lundberg, P.-A.; Mellstrom, D.; Oden, A.; Svanborg, A. **Factors Influencing Serum Free T_4 in 70-Year-Old Men. Implications for Reference Intervals in the Elderly.** *Scandinavian Journal of Clinical and Laboratory Investigation* 43(5): 401-413, September 1983.

Smoking was associated with significantly low body mass, but had no apparent effect on mean free thyroxine (T_4), the blood component best correlated with thyroid function, in a representative population of 460 males (aged 70). A procedure was tested for obtaining reference values based on careful clinical analysis of the sample with the background (1) that there are conflicting reports of age effects on thyroid function parameters, and (2) that recommended procedures for the selection of reference subjects in elderly populations may give unacceptably high exclusion rates. Serum free T_4 was determined by a ligand-analog technique, performed 5 years after the clinical study, and results were analyzed. The free T_4 concentration was significantly low in subjects with neoplastic disease or chronic bronchitis, and significantly high in subjects taking nonselective β-adrenergic blockers. Selective β_1-blockers had no apparent effect. Information on deaths proved to have no significance. The free T_4 concentration was found to be negatively correlated to body mass and body mass index. A procedure, which successively excludes clinically defined diagnostic groups with significantly low or high analyte concentration and/or body mass, may be used to obtain reference values for thyroid function and may reveal previously unknown relationships. (Auth. Abs. Mod.)

84–0047. Lundholm, M.; Rylander, R. **Work Related Symptoms Among Sewage Workers.** *British Journal of Industrial Medicine* 40(3): 325-329, August 1983.

Smokers in the sewage treatment plants had a significantly lower number of white blood cells than smokers in drinking water plants ($p<0.005$). No differences were found in the number of white blood cells or their distribution, or the number of platelets, between nonsmokers at the two types of plants. Sewage treatment plant employees (92 smokers and 107 nonsmokers) and drinking water plant employees (24 smokers and 17 nonsmokers) were interviewed for presence of specific medical symptoms (diarrhea and gastrointestinal symptoms, skin disorders) and evaluated for serum immunoglobulin concentrations, urine FDP concentrations, white blood cell counts, and airborne Gram-negative rods. Fatigue was attributed to work by a higher proportion of smokers than nonsmokers (not statistically significant). The proportion of workers reporting symptoms was approximately equal among smokers and nonsmokers at the sewage treatment plants, but nonsmokers reported a significantly higher proportion of symptoms than nonsmokers in drinking water plants.

84–0048. Malizia, E.; Andreucci, G.; Alfani, F.; Smeriglio, M.; Nicholai, P. **Acute Intoxication With Nicotine Alkaloids and Cannabinoids in Children From Ingestion of Cigarettes.** *Human Toxicology* 2(2): 315-316, 1983.

Data on 15 children (aged 9 months to 4 years) hospitalized following ingestion of tobacco cigarettes (10 cases) and cannabinoid joints or reefers (5 cases) shows that the four children who ingested two cigarettes were severely poisoned. All developed salivation, vomiting, diarrhea, tachypnea, tachycardia, and hypertension within 30 minutes, depressed respiration and cardiac arrhythmia within 40 minutes, followed by convulsions within 60 minutes after ingestion. The others, who ingested only a part of a cigarette, developed salivation and vomiting. Symptoms following reefer ingestion developed within 20 minutes, but only one case had a positive urine sample for nicotine and one for tetrahydrocannabinol.

84–0049. Marks, M. J.; Burch, J. B.; Collins, A. C. **Genetics of Nicotine Response in Four Inbred Strains of Mice.** *Journal of Pharmacology and Experimental Therapeutics* 226(1): 291-302, 1983.

The effects of nicotine on five behavioral and physiological measures were determined in four inbred mouse strains (BALB, C57BL, DBA, and C3H). In addition, the binding characteristics of nicotine and α-bungarotoxin (BTX), two ligands which appear to label different nicotinic receptors, were measured in seven discrete brain regions, and in the whole brain. Differences in response to nicotine were found among the four inbred strains. Whereas nicotine depressed open-field activity of BALB, C57BL, and DBA mice in a dose-dependent manner, low doses of nicotine increased locomotor activity in C3H mice. The doses of nicotine tested reduced Rotarod performance in DBA and C57BL mice, but not in C3H and BALB mice. All four strains displayed a dose-dependent decrease in body temperature after nicotine administration. The BALB mice were more sensitive than the C3H, whereas the effects on C57BL and DBA mice were intermediate. All four strains showed a transient increase in respiration only after a high (2.0 mg/kg) nicotine dose. Only the C3H mice exhibited enhanced startle response after auditory stimulation following nicotine administration. Differences in labeled nicotine binding among the seven brain regions were noted in each strain, but no differences among strains were observed. The TC_{50} values for inhibition of this binding by nicotine did not differ among brain regions within any strain or within any region among strains. Similarly, nicotine inhibited labeled BTX binding with equal potency in all brain regions of each of the four strains; however, the binding of this ligand was significantly lower in the midbrain and hippocampus of DBA mice than in these regions in the other three strains. Thus, genetic factors influence response to nicotine, but variation in response is not easily explained by differences in brain nicotinic receptors. (Auth. Abs. Mod.)

84–0050. Marlowe, C.; Waddell, W. J. **Affinity of (^{14}C)Nitrosopiperidine and Metabolites for Mouse Epithelial Tissues.** *Toxicology and Applied Pharmacology* 67(1): 110-116, January 1983.

Nitrosopiperidine (NPIP), a carcinogenic component of cigarette smoke condensate, was accumulated rapidly and exhibited prolonged retention in nasal and bronchial epithelium and in liver when examined by autoradiography. Male C57BL/6J mice given (^{14}C)NPIP and frozen at intervals of 0.1 to 24 hours were examined by whole-body autoradiography without thawing. The autoradiographs revealed intense localization of radioactivity at 6 minutes through 24 hours. At 24 hours, the most intense accumulation was in the epithelium of the bronchi, nose, salivary gland ducts, esophagus, and in the liver and Harder's gland. The results suggest that at least one metabolite of NPIP that localizes in the sites where tumors occur may be similar to a metabolite of N'-nitrosonornicotine. (Auth. Abs. Mod.)

84–0051. Martin, B. R.; Tripathi, H. L.; Aceto, M. D.; May, E. L. **Relationship of the Biodisposition of the Stereoisomers of Nicotine in the Central Nervous System to Their Pharmacological Actions.** *Journal of Pharmacology and Experimental Therapeutics* 226(1): 157-163, July 1983.

The stereoisomers of nicotine were evaluated for their effectiveness in producing antinociception and altering spontaneous activity and Rotarod performance in rats. (—)-Nicotine was found to be 6, 15, and 30 times more active than its unnatural enantiomer, (+)-nicotine, in the spontaneous activity, Rotarod, and antinociceptive tests, respectively. Biodispositional studies revealed that the time course of (—)-(^{3}H) nicotine closely paralleled the time course of effects on spontaneous activity and Rotarod performance but not antinociception, which suggested that multiple mechanisms were involved in the actions of nicotine. In addition, the distribution studies showed the brain and plasma levels of (—)-(^{3}H) nicotine were higher than those of (+)-(^{3}H) nicotine, which indicate that the pharmacological stereoselectivity of nicotine is less than originally determined. Finally, the greatest difference in the regional localization in brain of the stereoisomers was found in cerebral cortex, hippocampus, and corpus striatum. (Auth. Abs.)

84–0052. Mizobe, F.; Livett, B. G. **Nicotine Stimulates Secretion of Both Catecholamines and Acetylcholinesterase From Cultured Adrenal Chromaffin Cells.** *Journal of Neuroscience* 3(4): 871-876, April 1983.

Nicotine, a nicotinic agonist, was found to release both endogenous acetylcholinesterase (AChE) and catecholamines (CA) from monolayer cultures of purified bovine adrenal chromaffin cells; the muscarinic agonist methacholine did not. The concomitant release of CA and AChE evoked by nicotine was Ca^{++}-dependent with a correlation coefficient $r=0.82$ ($p<0.001$). The results show that adrenal chromaffin cells in vitro, a system free of splanchnic nerve elements, can still release AChE. The finding that concomitant release of AChE and CA occurs on exposure of the cells to nicotinic agonists suggests that released AChE may have a physiological role at neuroeffector junctions. (Auth. Abs. Mod.)

84–0053. Morgenstern, R.; Guthenberg, C.; Mannervik, B.; DePierre, J. W.; Ernster, L. **Benzo(*a*)pyrene Metabolism by Rat Liver Microsomes: Effects of Adding Purified Glutathione *S*-Transferases A, B, and C.** *Cancer Research* 42(10): 4215-4221, October 1982.

Addition of glutathione and isolated cytosolic glutathione *S*-transferases A, B, and C to rat liver microsomes metabolizing benzo(*a*)pyrene (BaP) resulted in up to 40 percent conjugate formation. Addition of glutathione alone resulted in the conjugation of 15 to 20 percent of the total metabolites of BaP, and this conjugation could be inhibited almost entirely by bromosulfophthalein (an inhibitor of glutathione *S*-transferases), indicating that it is catalyzed by the glutathione *S*-transferase present in microsomes. Addition of purified cytosolic glutathione *S*-transferases A, B, and C yielded about 30 to 40 percent conjugate formation. Analysis of metabolites by high pressure liquid chromatography demonstrated that the formation of the 4,5-diol of BaP was decreased by at least 80 percent by conjugation, and that 7,8-diol was also decreased significantly (40 to 60 percent). In addition, it was found that glutathione *S*-transferase B is capable of conjugating BaP 1,6- and 3,6-quinones. (Auth. Abs. Mod.)

84–0054. Perez-Reyes, M.; Di Guiseppi, S.; Mason, A. P.; Davis, K. H. **Passive Inhalation of Marihuana Smoke and Urinary Excretion of Cannabinoids.** *Clinical Pharmacology and Therapeutics* 34(1): 36-41, July 1983.

Results of three studies in which exposure to marihuana smoke was progressively increased found that of 80 urine samples collected, the drug levels in only two slightly exceeded the value of 20 ng/ml. In each study, four experienced marihuana users smoked marihuana cigarettes in the presence of two nonsmoking subjects. All were confined in a closed environment for 1 hour after the smoking began. All urine voided by the nonsmoking subjects was collected separately for 24 hours and analyzed for the presence of cannabinoids by the enzyme multiplied radioimmunoassay technique (EMIT) cannabinoid assay. Blood samples were drawn from one of the nonsmoking subjects in one of the studies, and Δ^9-tetrahydrocannabinol (THC) plasma concentrations were determined by radioimmunoassay. Minute but detectable concentrations were present in the plasma of the one subject from whom blood samples were obtained. In this subject, intravenous infusion of THC at the rate of 3.2 μg/min for 1 hour resulted in THC plasma concentrations identical to those during passive inhalation of marihuana smoke. In view of the findings, allegations that urine containing more than 50 ng/ml cannabinoids by the EMIT assay are the result of the passive inhalation of marihuana smoke are untenable or, at best, highly questionable. (Auth. Abs. Mod.)

84–0055. Pomerleau, O. F.; Fertig, J. B.; Seyler, L. E.; Jaffe, J. H. **Neuroendocrine Reactivity to Nicotine in Smokers.** *Psychopharmacology* 81(1): 61-67, August 1983.

Data suggest that nicotine stimulates release of certain central neuropeptides in proportion to its level in plasma. Habitual smokers smoked research cigarettes of known strength under controlled laboratory conditions while blood samples were withdrawn unobtrusively for subsequent biochemical analysis. To provide a measure that reflected total nicotine intake and total neurohormonal output, data were integrated over time. Subjects were relatively unresponsive in the low-nicotine (0.48 mg) condition. In the high-nicotine (2.87 mg) condition, there were significant positive correlations between integrated plasma nicotine and plasma arginine vasopressin ($r = +0.985$), its carrier protein neurophysin I ($r = +0.944$), and β-endorphin-β-lipotropin ($r = +0.977$), but not adrenocorticotropic hormone. Data from an experiment that used an extraction step to remove β-lipotropin corroborated the functional relationship between plasma nicotine and β-endorphin implied by the original findings. Taking into account recent research on the role of neuropeptides in the modulation of affective states, cognitive function, and other central nervous system activity, data strengthen the hypothesis that nicotine-stimulated neuropeptide release provides positive reinforcement for smoking. (Auth. Abs. Mod.)

84–0056. Potthoff, A. D.; Ellison, G.; Nelson, L. **Ethanol Intake Increases During Continuous Administration of Amphetamine and Nicotine, But Not Several Other Drugs.** *Pharmacology Biochemistry and Behavior* 18(4): 489-493, April 1983.

Nicotine- and d-amphetamine-treated rats were found to significantly increase their ethanol intake in tests done with a variety of psychoactive agents. Groups of rats, acclimated to drinking both water and 10 percent v/v ethanol, were implanted with a variety of slow-release devices containing d-amphetamine (d-amp), nicotine, caffeine, phencyclidine (PCP), secobarbital, LSD, mescaline, or haloperidol. Ethanol intake was elevated only during treatment with d-amp or nicotine; none of the other drugs affected ethanol consumption even though the amounts of all drugs released were pharmacologically sufficient to affect behavior. Nicotine-treated rats were not simply seeking calories provided by the ethanol solution, since nicotine treatment did not enhance intake of a distinctively flavored solution isocaloric to 10 percent ethanol. These results support a self-medication model of ethanol intake. (Auth. Abs. Mod.)

84–0057. Priola, D. V.; Curtis, M. B.; Anagnostelis, C.; Martinez, E. **Altered Nicotinic Sensitivity of AV Node in Surgically Denervated Canine Hearts.** *American Journal of Physiology* 245(1): H27-H32, July 1983.

Nicotinic supersensitivity, which followed cardiac denervation in dogs, was ascribed to increased responsiveness of the intrinsic cardiac nerves (ICN). The responses of normal and cardiac-denervated (DNV) dogs to acetylcholine (ACh) and nicotine (NIC) were examined to determine whether the ICN that modulate electrical conduction display denervation supersensitivity. Control ($n = 18$) and DNV ($n = 18$) animals were placed on cardiopulmonary bypass. Recordings of intra-atrial (P-A), intraventricular (H-V), and atrioventricular (AV) nodal (A-H) conduction times were made from the region of the His bundle. ACh (0.1-10 μg) was used to produce muscarinic stimulation, whereas NIC (0.1-400 μg) was employed to stimulate the ICN. All drugs were administered intracoronary. No supersensitivity to either ACh or NIC was seen in the data from the P-A or H-V intervals of the His electrogram in the DNV animals. However, this group displayed approximately a 10-fold increase in the negative dromotropic effect of NIC on the AV node compared with controls. No significant change in muscarinic sensitivity of the AV node was observed in the DNV animals. It is concluded that (1) no denervation supersensitivity of the ICN mediating effects on intra-atrial and intraventricular conduction occurs; (2) the AV node itself does not show muscarinic supersensitivity following extrinsic denervation; and (3) the ICN do display denervation supersensitivity as shown by a 10-fold increase in the effects of NIC on AV nodal conduction time. (Auth. Abs. Mod.)

84–0058. Rotenberg, K. S.; Adir, J. **Pharmacokinetics of Nicotine in Rats After Multiple-Cigarette Smoke Exposure.**

Toxicology and Applied Pharmacology 69(1): 1-11, June 15, 1983.

Multiple cigarette smoke inhalation appears to increase the extent but not the rate of nicotine absorption, decrease the rate and extent of its conversion to cotinine, and enhance the reduction of its polar *N*-oxide metabolite. The pharmacokinetics of nicotine and its major metabolites were evaluated in male rats exposed to smoke from a single cigarette every 8 hours for 14 days and to the smoke of a cigarette spiked with radiolabeled nicotine on the 15th day. Blood and urine samples were collected at timed intervals during the 10-minute smoke exposure period of the last cigarette and up to 48 hours thereafter. Nicotine, cotinine, and other polar metabolites were separated by thin-layer chromatography and quantified by liquid scintillation counting. Data were analyzed by computer fitting, and the derived pharmacokinetic parameters were compared with those observed after a single i.v. injection of nicotine and after a single cigarette smoke exposure. Results indicated that (1) the amount of nicotine absorbed from multiple cigarette smoke was approximately 10-fold greater than that absorbed from a single cigarette; (2) nicotine plasma levels did not decay monotonically but increased after the 5th hour; and (3) high plasma concentrations persisted for 30 hours. The rate and extent of the formation of cotinine, the major metabolite of nicotine, were decreased compared with values following a single cigarette smoke exposure. Urinary excretion tended to support the conclusion that the pharmacokinetic parameters of nicotine and its metabolites were altered upon multiple, compared with single, dose exposure. (Auth. Abs. Mod.)

84–0059. Russell, P. O.; Epstein, L. H.; Dickson, B. E. **Behavorial and Physiological Effects of Low-Nicotine Cigarettes During Rapid Smoking.** *Journal of Consulting and Clinical Psychology* 51(2): 312, 1983.

The effects of rapid smoking using medium-tar/nicotine or low-tar/nicotine cigarettes on physiological and behavioral responses were compared in a randomized, double-blind clinical trial. Results showed equivalent heart rate, blood pressure, and carbon monoxide levels after rapid smoking of either low- or medium-yield cigarettes. Behavorial topography measures differed significantly when low-nicotine cigarettes were smoked. Also, subjects who did remain abstinent at subsequent followup showed less change in rated aversiveness during rapid smoking than subjects who did not remain abstinent. (Auth Abs.)

84–0060. Russell, P. O.; Epstein, L. H.; Erickson, K. T. **Effects of Acute Exercise and Cigarette Smoking on Autonomic and Neuromuscular Responses to a Cognitive Stressor.** *Psychological Reports* 53(1): 199-206, August 1983.

Analysis showed smoking and exercise increased heart rate and skin conductance in 12 male sedentary smokers, while exercise reduced electromyographic activity. The effects of smoking and acute aerobic exercise as coping responses to stress were studied, and changes in heart rate, skin conductance, and electromyographic activity were assessed during the coping responses and a mental arithmetic stressor. No reliable effects of the coping responses on heart rate, skin conductance levels, or electromyographic changes to the stressor were observed. (Auth. Abs. Mod.)

84–0061. Sjogren, B.; Hedstrom, L.; Ulfvarson, U. **Urine Chromium as an Estimator of Air Exposure to Stainless Steel Welding Fumes.** *International Archives of Occupational and Environmental Health* 51(4): 347-354, 1983.

Of 53 male stainless steel welders (mean age 43), smokers (n=24) tended to have higher urine chromium concentrations than nonsmokers (n=29), although the difference was not statistically significant (p=0.08 for a test of distance between the regression lines). Chromium concentrations in air and postshift urine samples collected the same working day showed a linear relationship. Individual urine measurements are suggested as approximate estimators of current exposure, since postshift samples reflect chromium concentrations of both current and previous stainless steel welding fume exposure.

84–0062. Sjogren, B.; Lundberg, I.; Lidums, V. **Aluminium in the Blood and Urine of Industrially Exposed Workers.** *British Journal of Industrial Medicine* 40(3): 301-304, August 1983.

Urine aluminum concentrations were not associated with smoking habits, gender, or age in any of the workers exposed to aluminum through (1) electrolytic production of aluminum; (2) production of aluminum powder; (3) production of aluminum sulfate; or (4) aluminum welding. For comparison, a group of renal failure patients on dialysis also participated. The exposed group consisted of 81 males and 12 females (mean age 40 years), with an equal number of smokers and nonsmokers. The control group consisted of 49 males and 29 females (mean age 42 years, 40 percent smokers). Results showed that welders and workers making aluminum powder and aluminum sulphate had higher aluminum concentrations in blood and urine than nonexposed referents. Workers in electrolytic production had higher urine (but not blood) concentrations than controls. Thus, aluminum was absorbed by all industrially exposed workers. Blood concentrations were lower than those presumably associated with aluminum-induced encephalopathy in patients receiving dialysis. (Auth. Abs. Mod.)

84–0063. Sonnenfeld, G.; Hudgens, R. W. **Effect of Carcinogenic Components of Cigarette Smoke on *in Vivo* Production of Murine Interferon.** *Cancer Research* 43(10): 4720-4722, October 1983.

Treatment of mice with several potentially carcinogenic components of tobacco smoke inhibited in vitro interferon induction. Chemicals used included 4-aminobiphenyl and aniline-HCl (found in high concentrations in sidestream tobacco smoke); hydrazine sulfate (found in high concentrations in mainstream tobacco smoke); and 2-methylquinoline (found in intermediate concentrations in both sidestream and mainstream smoke). The chemicals were injected i.p. into mice, and α/β interferon was induced by i.v. injection of polyriboinosinic—polyribocytidylic acid. The interferon was induced either 2, 24, or 48 hours after treatment with the tobacco smoke components. Mice treated with 4-aminobiphenyl showed some depression of interferon production 2 hours after treatment, maximum inhibition of interferon induction 24 hours after treatment, and a return to control levels of interferon 48 hours after treatment. Mice treated with hydrazine sulfate showed maximum inhibition of interferon induction 24 hours after treatment but no effects at any other treatment time. These components were the most carcinogenic chemicals used. Treatment with aniline-HCl, a chemical whose carcinogenic potential is still debated, resulted in marginal depression of interferon induction 24 hours after treatment. 2-Methylquinoline, which had the lowest carcinogenic potential, had no effect on interferon induction. Efficacy of the chemical in inhibiting interferon induction was not influenced by predominance in mainstream or sidestream smoke. (Auth. Abs. Mod.)

84–0064. Stone, P. J.; Calore, J. D.; McGowan, S. E.; Bernardo, J.; Snider, G. L.; Franzblau, C. **Functional α_1-Protease Inhibitor in the Lower Respiratory Tract of Cigarette Smokers Is Not Decreased.** *Science* 221(4616): 1187-1189, September 16, 1983.

The mean level of functional α_1-protease inhibitor in the bronchoalveolar lavage (BAL) fluid of smokers (9 females and 12 males, average age 25 years) was found to be equal to or greater than that of nonsmokers (3 females and 13 males, average age 28 years), contradicting reports by other investigators. Binding and inactivation of isotopically labeled porcine pancreatic elastase and human neutrophil elastase by α_1-protease inhibitor were measured in fluid obtained by bronchoalveolar lavage. Increased elastase derived from pulmonary neutrophils, rather than decreased functional α_1-protease inhibitor, appears to be the main factor in the genesis of emphysema in smokers. The sedimented BAL cells from smokers averaged five times as many neutrophils and alveolar macrophages as were found in nonsmokers. If cells obtained by lavage are representative of cells within the lung, the elastase burden of smokers is clearly increased. (Auth. Abs. Mod.)

84–0065. Tang, B. K.; Kalow, W.; Inaba, T.; Kadar, D. **Variation in Amobarbital Metabolism: Evaluation of a Simplified Population Study.** *Clinical Pharmacology and Therapeutics* 34(2): 202-206, August 1983.

Urine analysis revealed a strong effect of smoking on amobarbital hydroxylation. Kinetic constants of amobarbital metabolism were established for 52 subjects (8 smokers, 8 to 22 cigarettes/day) on the basis of urinary analysis extending over several days, usually 96 hours. There was no evidence of effect of age or sex on any of the constants. C-Hydroxylation was induced by cigarette smoking as much as 100 percent, but glucosidation was not affected. Ethnicity (Caucasian or Oriental) also showed an influence. The differential effects of smoking on metabolite excretion suggest that either both cytochrome P-450 and P-448 monooxygenases are involved in C-hydroxylation of amobarbital, or the polynuclear aromatic hydrocarbons are not the only components of tobacco smoke involved in induction. (Auth. Abs. Mod.)

84–0066. Virshup, D. M.; Zinkham, W. H.; Sirota, R. L.; Caughey, W. S. **Unique Sensitivity of Hb Zuerich to Oxidative Injury by Phenazopyridine: Reversal of the Effects by Elevating Carboxyhemoglobin Levels In Vivo and In Vitro.** *American Journal of Hematology* 14(4): 315-324, 1983.

Data show that elevated levels of carboxyhemoglobin (COHb) in vivo and in vitro protect intact red cells from oxidative injury by phenazopyridine (PAP), a commonly used urinary tract analgesic. Incubation of whole blood from three asymptomatic Hb Zuerich subjects with PAP produced a moderate to marked increase in methemoglobin (MHb) and Heinz body formation accompanied by a slight to moderate decrease in levels of reduced glutathione. The rates of MHb formation were proportional to the concentration of PAP. In two subjects, the rates of formation of Heinz bodies and MHb were substantially higher than in the third subject. The two subjects with higher rates of MHb and Heinz body formation were nonsmokers with in vivo COHb levels ranging from 4 to 6 percent. The third subject, with low rates of MHb and Heinz body formation, was a smoker with in vivo COHb levels of from 15 to 18 percent. Increasing levels of COHb from 8.2 percent to 14.3 percent by the in vitro addition of carbon monoxide caused a marked reduction in the rate of Heinz body formation and a moderate decrease in MHb formation. Rates of MHb formation similar to those observed in normal red cells occurred at COHb percentages of 89.2 and 99.2. (Auth. Abs. Mod.)

84–0067. Waddell, W. J.; Marlowe, C. **Inhibition by Alcohols of the Localization of Radioactive Nitrosonornicotine in Sites of Tumor Formation.** *Science* 221(4605): 51-53, July 1, 1983.

Oral administration of alcohol to mice prior to injection of carbon-14-labeled nitrosonornicotine inhibited the localization of radioactivity in bronchial and salivary duct epithelium and in the liver. Adult male C57BL/6J mice were injected with $(2'\text{-}{}^{14}C)$nitrosonornicotine (0.12 to 0.19 μCi/g body weight, or 0.14 to 1.9 mg/kg). Some of the mice had been treated 20 minutes earlier with ethanol, *n*-butanol, or *t*-butanol (0.02 ml/g body weight). In pretreated mice, autoradiography showed reduced localization of radioactivity in salivary duct and bronchial epithelium, and in both the periportal and central areas of the liver, but not in the nasal epithelium or esophagus. It is suggested that these alcohols may selectively inhibit tumor formation in sites of reduced radioactivity localization.

84–0068. Wagner, V.; Skokanova, K.; Wagnerova, M.; Heribanova, A.; Riha, M. **Indicators of Humoral Immunity in Smokers and Nonsmokers Working Underground.** *Journal of Hygiene, Epidemiology, Microbiology and Immunology* 27(2): 129-142, 1983.

Long-term employment in shale mines and smoking had interactive effects on immune system status in underground miners. Underground shale miners (148 smokers and 67 nonsmokers) were examined for levels of immunoglobulins in sera (IgG, IgA, IgM) and saliva (sIgA), transferrin, alpha 2-macroglobulin (A 2M) and lysozyme levels in sera (LYS) and saliva (sLYS). A control group of 95 healthy men was selected, matched for age. The mine environment was characterized by ionizing radiation, due to a geological configuration of the rock. The decline of IgG, IgA, and sLYS levels in smokers was significantly deeper than in nonsmokers and controls, while levels of sIgA and serum LYS increased both in smokers and nonsmokers. The change in the serum LYS and A 2M levels in miners did not depend on the smoking habits, except of A 2M levels in miners employed for more than 21 years. Generally, differences were less pronounced in younger miners and tended to increase with the increasing length of work underground. (Auth. Abs. Mod.)

84–0069. Watanabe, T.; Endo, A.; Kumai, M.; Ikeda, M. **Chromosome Aberrations and Sister Chromatid Exchanges in Styrene-Exposed Workers With Reference to Their Smoking Habits.** *Environmental Mutagenesis* 5(3): 299-309, 1983.

While exposure to styrene did not affect sister-chromatid exchange (SCE) frequencies in cultured lymphocytes, styrene-exposed smokers tended to have higher SCE frequencies than exposed nonsmokers. The incidences of chromosome aberrations and the frequencies of SCE were investigated in cultured lymphocytes of 18 styrene-exposed workers (13 smokers) and compared with 6 controls (3 smokers). There was a marginal increase in the incidence of structural chromosomal aberrations in first-division metaphases in the styrene-exposed workers compared with the nonexposed controls. However, there was no difference in SCE frequencies. Cell proliferation was also inhibited in styrene-exposed workers (both smokers and nonsmokers) and control smokers. It is suggested that the SCE frequency in such styrene-exposed workers is influenced by individual smoking habits and both may act synergistically to damage certain properties of lymphocytes, resulting in increased SCE frequencies and cell cycle inhibition. (Auth. Abs. Mod.)

84–0070. Wiersma, D. A.; Roth, R. A. **Clearance of Benzo(*a*)pyrene by Isolated Rat Liver and Lung: Alterations in Perfusion and Metabolic Capacity.** *Journal of Pharmacology and Experimental Therapeutics* 225(1): 121-125, April 1983.

Results indicate that flow and metabolic capacity are important determinants of both hepatic and pulmonary clearance of xenobiotic compounds, and suggest that alterations in either factor may affect the relative roles of these organs in total body disposition of xenobiotic compounds. The ability of isolated rat livers and lungs to clear circulating benzo(*a*)pyrene (BaP) was examined under conditions of altered perfusion and pretreatment with 3-methylcholanthrene (3-MC). In isolated organs from control rats, pulmonary BaP clearance was low (approximately 1 ml/min) and independent of flow between 10 and 45 ml/min, whereas hepatic clearance was greater and flow-dependent, increasing from 3.2±0.2 ml/min at a flow of 7 ml/min to 13.2±0.7 ml/min at a flow of 20 ml/min. 3-MC pretreatment markedly increased the aryl hydrocarbon hydroxylase activity of broken cell preparations from both organs. While BaP clearance was not increased in isolated livers by 3-MC pretreatment, clearance did increase and become flow-dependent in isolated lungs of 3-MC-pretreated rats. When organs from these animals were perfused at flows occurring in vivo, hepatic and pulmonary clearance were about equal. (Auth. Abs. Mod.)

84–0071. Williams, M. K.; Walford, J.; King, E. **Blood Lead and the Symptoms of Lead Absorption.** *British Journal of Industrial Medicine* 40(3): 285-292, August 1983.

Blood lead determination and/or erythrocyte protoporphyrin (EPP) measurement, plus questionnaire data (modified Cornell medical index), showed that heavy smokers (>20 cigarettes/day) had the highest mean score in each symptom category (physical, psychological, potentially lead-induced, nervous system, genitourinary, fatigue). Subjects were 527 male lead acid battery and plastics employees, evaluated by age and smoking habits. The mean score of nonsmokers for psychological symptoms was slightly lower than that of heavy smokers. Data indicated little difference in the percentage of heavy smokers in the blood lead groups (<40 μg/100 ml (1.93 μmol/l); ≥40 μg but <60 μg (2.90 μmol/l); or ≥60 μg), while variation was noted in the non-, light-, and ex-smoking categories. Excluding the highest log EPP group, where numbers were small, the percentages of both nonsmokers and heavy smokers decreased as log EPP increased. While results could be biased due to participants' previous knowledge of symptoms associated with lead exposure, the possibility of overexposure must be considered.

84–0072. Wulf, H. C.; Husum, B.; Niebuhr, E. **Sister Chromatid Exchanges in Smokers of High-Tar Cigarettes, Low-Tar Cigarettes, Cheroots and Pipe Tobacco.** *Hereditas* 98(2): 225-228, 1983.

Analysis of sister-chromatid exchange (SCE) in 189 smokers of different tobacco types and 60 nonsmokers showed that all the smokers had significantly elevated SCE values ($p < 0.01$), regardless of type of tobacco smoked. Smokers of filtered high-tar cigarettes ($n = 64$) had 9.47±0.15 SCE/cell (mean value and standard error of the mean). Smokers of unfiltered high-tar cigarettes ($n = 42$) had 9.19±0.19 SCE/cell, and smokers of filtered low-tar cigarettes ($n = 23$) had 8.97±0.24 SCE/cell. Consumption ranged from 15 to 17 cigarettes/day, and

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differences in SCE between the smoking groups were not statistically significant. The 60 nonsmokers had 8.25±0.13 SCE/cell, while the 30 cigar smokers (average daily consumption 8.3 cigars) had 10.47±0.38 SCE/cell, significantly higher than the nonsmoking group ($p<0.001$). The 30 pipe smokers (daily tobacco consumption 13.7 g) had an SCE count of 9.60±0.23, also significantly higher than the nonsmoking group ($p<0.001$). Data showed no relationship between condensate and filter in cigarettes and SCE. (Auth. Abs. Mod.)

84–0073. Zabik, J. E.; Roache, J. D.; Johnson, W.; Nash, J. F., Jr.; Maickel, R. P. **Dependence Liability of Caffeine, Cocaine, Nicotine and Phencyclidine.** *Research Communications in Substances of Abuse* 4(1): 93-96, 1983.

A test system of deprivation-induced fluid consumption in rats was used to examine the effects of caffeine, cocaine, nicotine, and phencyclidine. None of the drugs stimulated the consummatory behavior of the animals, but all four caused dose-related decreases in water intake. The extrapolated dosage required for a 25 percent reduction in fluid consumption ranged from 4.69 μmoles/kg for nicotine to >412 μmoles/kg for caffeine. These results do not support physical dependence liability for these agents in humans. (Auth. Abs. Mod.)

84–0074. Zasiow, M. C.; Clark, R. A.; Stone, P. J.; Calore, J. D.; Snider, G. L.; Franzblau, C. **Human Neutrophil Elastase Does Not Bind to Alpha$_1$-Protease Inhibitor That Has Been Exposed to Activated Human Neutrophils.** *American Review of Respiratory Disease* 128(3): 434-439, September 1983.

While smoking may increase the protease burden of the lung to produce emphysema regardless of the functional status of α_1-protease inhibitor (α_1-PI), the frequent development of emphysema in smokers with homozygous deficiency of α_1-PI demonstrates that the quantity of α_1-PI is clinically important. The effect of leukocyte-derived oxidants on the elastase-inhibitory capacity of α_1-PI was examined in an in vitro system using cells and purified proteins from human sources. The exposure of α_1-PI to the myeloperoxidase-hydrogen peroxide-halide system resulted in a nearly complete loss of its ability to bind and inactivate purified human neutrophil elastase. A similar loss of binding to and inactivation of human neutrophil elastase was observed on exposure of α_1-PI to human neutrophils in the presence of a halide and the neutrophil-activating agent, phorbol myristate acetate. This loss of elastase binding activity was abrogated by the addition of azide or catalase but not superoxide dismutase or heated catalase. The data suggest oxidative inactivation of α_1-PI by secreted myeloperoxidase and hydrogen peroxide. Thus, the reported effects of leukocyte oxidants, especially the myeloperoxidase system, on α_1-PI have been confirmed using the most pathophysiologically relevant protease, human neutrophil elastase, as the test enzyme. Emphysema may result when the normal remodeling function of neutrophils is triggered either by cigarette smoke or by events such as secretion of chemotactic factors by macrophages. (Auth. Abs. Mod.)

See also, **84–0108**

MORTALITY AND MORBIDITY

84–0075. Blair, A.; Berney, B. W.; Heid, M. F.; White, D. W. **Causes of Death Among Workers in the Tobacco Industry.** *Archives of Environmental Health* 38(4): 223-228, July-August 1983.

Obituary listings (1957 to 1978) for 2,709 tobacco industry workers (1,003 white males, 789 black males, 517 white females, 400 black females) showed that mortality due to cancer of the colon was slightly elevated for each race—sex group (proportionate mortality ratios (PMRs) from 129 to 163). Although unexplained, this result resembles the excess of cancer of the digestive system previously reported among cigarette manufacturers. Excess deaths from cancer of the lung occurred only among white females and may represent chance findings. Among nonneoplastic causes of death, PMRs for arteriosclerotic heart disease, motor vehicle accidents, and suicides were high among most race—sex groups, while PMRs for respiratory disease and cirrhosis of the liver were low. While information on important lifestyle characteristics (such as tobacco and alcohol consumption) was not available, data on smoking and mortality among World War I veterans suggest that a larger proportion of workers in the tobacco industry smoked cigarettes than workers from other industries (51 percent vs. 40 percent). (Auth. Abs. Mod.)

84–0076. Bush, T. L.; Comstock, G. W. **Smoking and Cardiovascular Mortality in Women.** *American Journal of Epidemiology* 118(4): 480-488, October 1983.

Data associated cigarette smoking at younger ages with increased mortality from all causes and from total and sudden arteriosclerotic disease-related mortality. Smoking status and sociodemographic characteristics were recorded for 23,572 white women (aged 25 to 74 years) in a Washington County, Maryland, census conducted in 1963. Deaths from all causes, from total and sudden arteriosclerotic heart disease, and with stroke were recorded for the next 12 years. Smoking-specific mortality rates for women aged 25 to 44, 45 to 64, and 65 to 74 years at entry were calculated after adjustment for the effects of marital status, education, housing quality, and frequency of church attendance. Among women in the oldest age group, smoking was not related to mortality. For women in the two younger age groups, the risks of dying from any cause and from arteriosclerotic heart disease (total and sudden) were positively associated with cigarette smoking. For all arteriosclerotic heart disease deaths, the relative risks associated with smoking more than 20 cigarettes/day were 3.6 and 2.2 for women aged 25 to 44 and 45 to 64, respectively; for sudden deaths from arteriosclerotic heart disease, the relative risks were 6.5 and 2.7. The risk of dying with stroke was not associated with cigarette smoking. (Auth. Abs. Mod.)

84–0077. Garn, S. M.; Hawthorne, V. M.; Pilkington, J. J.; Pesick, S. D. **Fatness and Mortality in the West of Scotland.** *American Journal of Clinical Nutrition* 38(2): 313-319, August 1983.

Mortality data over 16 years from 2,381 males (1,322 smokers) aged 45 to 75 from the West of Scotland showed that in each smoking category, the lean had a higher mortality rate than the obese. At all body weight levels, smokers had a higher mortality than the nonsmokers. The subjects were stratified into nonsmokers, moderate ($\leq$ 10 cigarettes/day), and heavy ($\geq$ 10 cigarettes/day) smokers. It is noted that for all five measures of obesity, respiratory cancer declined from the lean to the obese while cardiovascular mortality increased. The notion of an "ideal weight" is challenged, and the importance of differentiation for smoking status in weight—mortality research is emphasized.

84–0078. Herman, B.; Schmitz, P.I.M.; Leyten, A.C.M.; Van Luijk, J. H.; Frenken, C.W.G.M.; Op de Coul, A.A.W.; Schulte, B.P.M. **Multivariate Logistic Analysis of Risk Factors for Stroke in Tilburg, the Netherlands.** *American Journal of Epidemiology* 118(4): 514-525, October 1983.

Cigarette smoking was not associated with the risk of stroke in a case—control study that evaluated personal data, health-related behavior, and medical history as stroke risk factors. Relative risk (RR) associated with smoking exposure (number of cigarettes smoked/day multiplied by number of years smoking) of <25 was 1.00, while RRs for smoking exposures of 25 to 399, 400 to 699, and ≥ 700 were 1.26, 1.43, and 1.47, respectively. Study subjects included 132 stroke patients and 239 age- and sex-matched control patients interviewed at two city hospitals in Tilburg, the Netherlands. To assess joint effects and possible interactions, and to control for multiple confounding factors, a series of multivariate logistic models for matched data were studied. From this analysis, it appeared that hypertension, acute myocardial infarction, cardiac arrhythmias, transient cerebral ischemic attacks, obesity, physical activity during leisure time, education of head of household, and Rhesus factor were all significant stroke risk factors. These risk determinants demonstrated a general multiplicative effect; however, the influence of some variables on stroke risk was not constant with age and sex. The relationship of diabetes mellitus to stroke slightly decreased and became nonsignificant after adjustment for factors besides age and sex. In addition to cigarette smoking, stroke risk was not associated with alcohol consumption, family history of stroke and related disorders, marital status, or ABO blood typing. (Auth. Abs. Mod.)

84–0079. Holden, C. **Can Smoking Explain Ultimate Gender Gap?** *Science* 221(4615): 1034, September 9, 1983.

Comments are presented regarding a study (*Public Health Reports* 98(4): 343-349, July-August 1983), which concluded that virtually all the increase in the difference between male and female longevity since 1930 is attributable to the effects of cigarette smoking. The study is criticized for its low response rate and small number of participants, and because the 3-year time span was insufficient to establish mortality rates. Other studies have shown that nonsmoking women outlive nonsmoking men by 2 to 4 years. It is concluded that smoking accounts for about half the difference in male and female longevity. See also 84-0082.

84–0080. King, D. R.; Smith, A. H.; Salter, D. M. **Mortality Attributable to Smoking in New Zealand.** *New Zealand Medical Journal* 96(728): 195-199, March 23, 1983.

Relative risks (RRs) for mortality due to various smoking-related diseases were derived from a literature review of the epidemiology of smoking-related illness. RRs were used, with New Zealand 1976 census data on smoking habits and National Health Statistics mortality data, to derive the proportion of deaths attributable to cigarette smoking. Results indicate that about 15 percent of deaths in New Zealand (3,693 annually) are smoking-related (19 percent of all male deaths and 8 percent of all female deaths in 1976). (Auth. Abs.)

84–0081. Metzner, H. L.; Carman, W. J.; House, J. **Health Practices, Risk Factors, and Chronic Disease in Tecumseh.** *Preventive Medicine* 12(4): 491-507, July 1983.

Never having smoked, one of seven health practices associated with a self-report index of good health and lower mortality in the Alameda County studies, was associated with chronic bronchitis (negatively) and forced expiratory volume in 1 sec (FEV$_1$). The seven practices were related to several measures of health in a cohort of 880 men (82 never smokers) and 973 women (237 never smokers) aged 35 to 69, who were participants in the Tecumseh Community Health Study. The health measures were prevalence of coronary heart disease (CHD), hypertension, and chronic bronchitis, and levels of five risk factors (systolic and diastolic blood pressures, serum total cholesterol, blood glucose, and FEV$_1$). Four of the practices (desirable relative weight, physical activity, never having smoked, and moderate or no alcohol drinking) were associated significantly with one or more of the health measures. Eating breakfast, limited between-meal eating, and sleeping 7 or 8 hours were not related to the health measures. Combining all seven health practices into an index produced a measure that was more strongly associated negatively with CHD prevalence than was any one of the component practices. However, the index was no more strongly associated with any of the other health measures than were the relevant separate practices. Desirable weight was more highly associated (negatively) with hypertension and blood pressure than the index. (Auth. Abs. Mod.)

84–0082. Miller, G. H.; Gerstein, D. R. **The Life Expectancy of Nonsmoking Men and Women.** *Public Health Reports* 98(4): 343-349, July-August 1983.

Nearly identical life expectancy figures for nonsmoking men and women of parallel age suggest that (1) dissimilar rates of cigarette smoking may explain the difference in male vs. female longevity, and (2) present longevity differences between men and women will disappear. Retrospective data for 2,195 deceased lifetime nonsmokers were combined with telephone survey data on lifetime smoking habits (with careful distinctions between former and never smokers) of 3,916 residents of Erie County, Pennsylvania, aged 30 or more years. Results imply that sex role behavior among males (i.e., excessive smoking, alcohol consumption, disdain for minor medical assistance, and suicidal and accident-risk tendencies) affect health, morbidity, and longevity, and that female health and morbidity will suffer as their behavior trends become increasingly similar to trends among males. Smoking is increasing among females, and teenage rates are higher for females than for males. When women who have smoked as much as men reach the later decades of life, their lives may also be shortened, eliminating present differences in longevity. Life tables for nonsmokers by sex and age group are provided, and analytical methods are explained.

84–0083. Pearce, N. E.; Davis, P. B.; Smith, A. H.; Foster, F. H. **Mortality and Social Class in New Zealand. I: Overall Male Mortality.** *New Zealand Medical Journal* 96(730): 281-285, April 27, 1983.

Lifestyle factors such as smoking, exercise, and diet are involved in the higher mortality rates among lower social classes, as are access to and utilization of health services and occupational hazards. Two different systems of social class definition were used to investigate male mortality rates in New Zealand. In each case it is found that the lower social classes have mortality rates significantly higher than those of the upper social classes, with the mortality rate of the lowest class being approximately twice that of the highest class on a six-category scale. The relative risk is higher in the younger age groups. Smoking incidence was lowest in the professions (average 27 percent) and highest in the less skilled occupations (average >50 percent). The smoking habit itself, however, is only a partial explanation, since it is still unknown why manual workers smoke more and find it more difficult to quit. (Auth. Abs. Mod.)

84–0084. Polednak, A. P.; Stehney, A. F.; Lucas, H. F. **Mortality Among Male Workers at a Thorium-Processing Plant.** *Health Physics* 44(Supplement 1): 239-251, 1983.

While the proportion of smokers was probably greater than data show due to disproportionate smoking-related mortality, information on the smoking habits of a sample ($n = 294$) of 592 survivors who had highest occupational exposure to thorium and thoron suggests that smoking was a factor in excess mortality. The standardized mortality ratio (SMR) for pancreatic cancer was significantly elevated (4.13, 95 percent confidence limit (CL) 1.34 and 9.63), while the SMR for lung cancer was 1.68 (95 percent CL 0.81 and 3.09), and the SMR for respiratory diseases was 1.20 (95 percent CL 0.52 and 2.37). Overall mortality data for a cohort of 3,039 males occupationally exposed to thorium between 1940 and 1973 show high SMRs for cancer of the lung (1.44, CL 0.98 and 2.02), pancreas (2.01, CL 0.92 and 3.82), and respiratory diseases (1.31, CL 0.92 and 1.83).

See also, **84–0170**

NEOPLASTIC DISEASES

84–0085. Ames, R. G. **Gastric Cancer and Coal Mine Dust Exposure. A Case—Control Study.** *Cancer* 52(7): 1346-1350, October 1, 1983.

A statistically elevated gastric cancer risk exists for coal miners who have prolonged exposure to both coal mine dust and cigarette smoke. Results of a case—control study (46 cases of mortality from gastric cancer matched by age with 138 controls) show an odds ratio (OR) of 3.10 for current smokers, and an OR of 3.52 for 30 or more years of smoking. OR (in 1-for-1 matched-case analysis) for more than 30 years of smoking is 6.00 against other cancer; and 3.00 against noncancer, nonaccident controls. Failure of ORs to achieve statistical significance at the 0.05 p level is attributed to the small sample sizes involved. While elevated gastric cancer risk for coal mine dust exposure is limited to coal miners who smoke cigarettes, cigarette smoking duration and continuance appear to be more important than quantity smoked/day. Furthermore, smoking status did not modify the risk associated with coal workers' pneumoconiosis, which was not found to be a risk factor for gastric cancer. Further study of the mechanism producing the interactive effect between coal mine dust exposure, cigarette smoking, and gastric cancer is suggested.

84–0086. Baimakanova, S. Sh.; Kairkenov, S. K. **Faktory Riska v Formirovanii "Grupp Riska" po Raku Gortani. [Risk Factors in Forming the "Risk Groups" of Laryngeal Cancer.]** *Zhurnal Ushnykh, Nosovykh I Gorlovykh Boleznei* 6: 53-56, 1982, Russian.

Epidemiologic studies revealed seven risk factors for laryngeal cancer: duration and intensity of smoking, sex, age, occupational hazards and length of employment, and laryngeal diseases. A plan, which proposed four levels of risk, was formulated to assess the risk of laryngeal cancer development. Periodicity of prophylactic examinations was determined in the four different risk groups. A retrospective study of 311 case histories revealed that III and IV grades of risk were present in 79.5 percent of laryngeal cancer patients. It is proposed that the plan be used in the course of prophylactic examinations. (Auth. Abs. Mod.)

84–0087. Bassett, M. R.; Dobie, R. A. **Patterns of Nutritional Deficiency in Head and Neck Cancer.** *Otolaryngology—Head and Neck Surgery* 91(2): 119-125, April 1983.

While all 50 head and neck cancer patients assessed for nutritional status were current or ex-smokers (average pack-years 66±32, three pipe smokers), data showed no correlation between poor nutritional assessment score and smoking or the clinical variables of age, sex, patient food preparation, alcohol consumption, or hospital type. Excess tobacco and alcohol consumption, poor dental hygiene, and poor nutrition often preceded clinical expression of malignancy, and the best predictor of impaired nutritional status was the recent diet as described by the patient. General diets predicted good scores, whereas diets restricted to soft or liquid foods predicted fair or poor scores, with overall accuracy of 72 percent. Pharyngeal tumors and higher stage tumors of any location were more often associated with fair or poor nutritional assessment scores. Anthropometric measurements, skin tests, laboratory assessment of visceral and somatic proteins, and questionnaires showed that 40 percent of the patients had good nutritional status, 20 percent had fair nutrition, and 40 percent had poor status, with elements of both protein—calorie depletion and visceral protein malnutrition.

84–0088. Beg, M.H.A.; Rehman, A.; Malik, S.; Qayum, A. **Pattern of Malignant Tumours in Otolaryngology in Karachi.** *Journal of the Pakistan Medical Association* 33(5): 110-114, May 1983.

Between 1976 and 1981, 163 malignant lesions of the pharynx, esophagus, and oral cavity were examined in workers and their families at the KV Social Security SITE hospital. Indian male emigrants with specific chewing habits, aged 41 and 50 years, were most often affected. Incidence was 10 cases in 100,000 secured workers/year. Betel and tobacco chewing habits were present in 128 Indian emigrant cases; 32 also smoked cigarettes. Only 18 cases did not use tobacco in some form. The cheek mucosa, tongue, vallecula, and the pyriform fossa, which are in direct contact with tobacco, accounted for a majority of cases. Histopathology revealed 84 percent of the tumors to be squamous cell carcinoma, and TNM staging classified most to be in the advanced stage. No evidence was found to label submucous fibrosis as a premalignant condition. (Auth. Abs. Mod.)

84–0089. Bernfeld, P.; Homburger, F.; Soto, E. **Subchronic Cigarette Smoke Inhalation Studies in Inbred Syrian Golden Hamsters That Develop Laryngeal Carcinoma Upon Chronic Exposure.** *Journal of the National Cancer Institute* 71(3): 619-623, September 1983.

The incidence and severity of laryngeal hyperplasia increased in inbred BIO 15.16 Syrian golden hamsters exposed for 6 to 20 weeks to smoke from three types of experimental cigarettes. Two laryngeal papillomas appeared, alveolar macrophages became more frequent and aggregated, and hyperplasia of terminal bronchiolar epithelium occurred. This subchronic response of hamsters to smoke markedly differed for the three types of cigarettes. Statistical evaluation of the data by log linear models proved these differences to be significant. At equal doses of smoke, the most severe response was caused by an all-tobacco cigarette. The weakest subchronic effects, next to those seen in the negative control group, were elicited by smoke from a cellulose-derived tobacco supplement. The effects of smoke from a 1:1 blend of the two smoking materials were intermediate. The severity of the subchronic response of the respiratory tract paralleled the extent of malignant transformations of the larynx previously observed in the same animal model with the same three types of cigarettes in chronic inhalation studies. (Auth. Abs.)

84–0090. Cavanaugh, D. G. **Lung Cancer and Its Association with Other Primary Malignancies.** *Military Medicine* 148: 331-332, April 1983.

Over 90 percent of lung cancer patients with other primary malignancies were smokers (all 19 men, 7 of 9 women). A total of 610 cases of lung cancer was identified over a 27-year period at the William Beaumont Army Medical Center. Lung cancer presented first in 11 of cases of multiple primaries. Average age was 52 in males, 54 in females; average time between first and second primary was 1.5 years for both. When lung cancer appeared as a second primary (17 cases), the average age for the initial primary was 10 years earlier for females compared with males (53 vs. 42 years). The average time between the appearance of the initial primary and lung cancer was 2 years longer in females compared with males. Average pack-years smoked for lung cancer as first primary were 50 for males and 27 for females; however, for lung cancer as second primary, pack-years smoked were 42 for males and 50 for females.

84–0091. Correa, P.; Johnson, W. D. **Cancer and Lifestyle in Louisiana.** *Journal of the Louisiana State Medical Society* 135(3): 4-6, March 1983.

While no definitive causal relationship is inferred, smoking and respiratory cancer incidence were higher in four southern compared with five northern counties in Louisiana. Questionnaire responses showed that 38.7 percent of the white and 54.0 percent of the black males in the south were smokers compared with 34.7 percent of the white and 46.9 percent of the black males from the north. The number using nonfiltered cigarettes, the proportion that started smoking before age 20, and (in whites) the excess of parents who smoked, were all higher in the south. Occupational differences were also found; oil and shipbuilding industries in the south employed one-third of the white labor force while farming and sawmill operations were predominant in the north. While an association between lung cancer and certain occupations has been suggested, it is recommended that any interpretation of such correlations take into account existing differences in smoking habits between occupational categories.

84–0092. Cowles, S. R. **Cancer of the Larynx: Occupational and Environmental Associations.** *Southern Medical Journal* 76(7): 894-898, July 1983.

Smoking and the combination of smoking and alcohol consumption are the two major risk factors for cancer of the larynx. Data have shown that the risk in smokers is from 3 to 39 times that of nonsmokers. The decline in male—female ratios (14.9 to 1.0 in 1956 compared with 4.6 to 1.0 in 1974) is consistent with increasing prevalence of smoking among women. Ex-smokers who have not smoked for 10 to 15 years are at half the risk of current smokers, while smokers of filtered (vs. unfiltered) cigarettes are at less risk. Relative risks (RR) associated with heavy alcohol consumption and heavy smoking combined are 50 percent greater than would be expected from the addictive effects of both factors, which are also important in development of double primary cancers of the head and neck. For both smoking and alcohol consumption, RR is greater among younger age groups. Laryngeal cancer patients who smoked were an average of 15 years younger than nonsmokers (aged 58 compared with age 73, respectively), while an Irish study showed that RR for heavy smokers under age 60 was 18 times that of nonsmokers. In France, unfiltered black tobacco cigarettes, smoked without inhaling, have been linked to increased risk for laryngeal cancer. Additional environmental risk factors associated with laryngeal cancer include chewing betel nuts, "pan" use in India, and chewing miang in Thailand. Data also showed a mild synergism between smoking and asbestos exposure in laryngeal cancer development among shipyard workers, while a case—control study (n=47) in Washington State, which found an insignificant correlation with asbestos exposure, showed a strong dose—response effect between smoking and cancer of the larynx; only one patient was a nonsmoker.

84–0093. Daniel, F. B.; Schut, H.A.J.; Sandwisch, D. W.; Schenck, K. M.; Hoffmann, C. O.; Patrick, J. R.; Stoner, G. D. **Interspecies Comparisons of Benzo(*a*)pyrene Metabolism and DNA-Adduct Formation in Cultured Human and Animal Bladder and Tracheobronchial Tissues.** *Cancer Research* 43(10): 4723-4729, October 1983.

Cultured bladder and tracheobronchial explants from human, monkey, dog, hamster, and rat were used to study the metabolism, covalent binding to DNA, and DNA adduct formation of (^3H)benzo(*a*)pyrene (BaP). Both organs from all species formed large amounts of polar material and small amounts of organic solvent-extractable (unconjugated) metabolites, with the balance being unmetabolized BaP. Unconjugated BaP metabolites showed qualitatively similar metabolism in the various species. Relative proportions of metabolites differed both between organs and between species. Total metabolism was highest in human and lowest in rat tissues. Also, covalent binding was significantly higher in human bladder and bronchus than in the corresponding animal tissues. At least four BaP/DNA adducts could be detected in both tissues from all the species. The distribution of BaP/DNA adducts is specific and reproducible for each species. In most cases, the most abundant adduct was that derived from the most carcinogenic of the known BaP metabolites. (Auth. Abs. Mod.)

84–0094. Deutsch-Wenzel, R. P.; Brune, H.; Grimmer, G.; Dettbarn, G.; Misfeld, J. **Experimental Studies in Rat Lungs on the Carcinogenicity and Dose-Response Relationships of Eight Frequently Occurring Environmental Polycyclic Aromatic Hydrocarbons.** *Journal of the National Cancer Institute* 71(3): 539-544, September 1983.

The biologic activity of eight highly purified polycyclic aromatic hydrocarbons (PAH), widely distributed in the human environment and in cigarette smoke condensate, was tested in the respiratory tracts of rats. The lung implantation method was used in 3-month-old female OM rats. Histologic and mathematical evaluations indicated that the investigated compounds had distinct carcinogenic potencies. A dose—response relationship was obtained for benzo(*a*)pyrene (BaP), anthanthrene (ANT), benzo(*b*)fluoranthene (BbF), and benzo(*k*)fluoranthene (BkF). Benzo(*e*)pyrene and benzo(*ghi*)perylene showed no tumor-producing effect in this system when given at doses of 5 mg. After probit analysis of the results, the carcinogenic potencies of PAH investigated in the lung implantation model rank as follows: BaP, 1.00; ANT, 0.19; BbF, 0.11; and BkF, 0.03. (Auth. Abs. Mod.)

84–0095. Eddy, D. M. **Finding Cancer in Asymptomatic People. Estimating the Benefits, Costs and Risks.** *Cancer* 51(12): 2440-2445, June 15, 1983.

It is estimated that the reduction in lung cancer mortality achievable by annual chest X-rays is approximately 2 percent, while the risk of developing lung cancer is about 15 percent in smokers over 40 years of age. Annual X-rays for smokers over age 40 are therefore not recommended since the costs and risks (smokers tend to feel protected by the annual exam) outweigh any potential benefits. All trials to date have failed to show that even intensive screening with chest X-rays and sputum cytology as frequently as three times a year significantly reduces lung cancer mortality. There are no absolute criteria for determining the effect of early detection on cancer mortality, but the best evidence is provided by randomized controlled trials that use mortality as an endpoint. The decision to recommend a test for a particular person must take into account the strength of the evidence that early detection reduces mortality, the expected amount of the reduction, the chance the person will ever get the cancer, and the anticipated costs and risks. In addition to screening for lung cancer, two specific examples are discussed: breast self-examination and mammography in women younger than 50 years of age. (Auth. Abs. Mod.)

84–0096. Feld, R. **Symposium on the Diagnosis and Treatment of Common Cancers. 1. Lung Cancer—1983.** *Canadian Journal of Surgery* 26(3): 266-268, May 1983.

In the United States, cigarette smoking is the main cause of lung cancer, now the most common site of cancer in men (22 percent of incidence and 34 percent of deaths) and the fourth most frequent site among women (9 percent of incidence and 16 percent of deaths). Data for Canada are similar. Lung cancer incidence is increasing rapidly, particularly among women, and increased prevalence in households exposed to cigarette smoke suggests a passive smoking effect. Diagnosis, staging, and treatment techniques (operation, radiotherapy, chemotherapy, and adjuvant therapy) are described. New developments, including recently discovered drugs that may be of value, and controversies over treatment regimens that have not been resolved, are outlined.

84–0097. Fischer, D. S. **Answers to Questions on Occult Neoplasms.** *Hospital Medicine* 19(9): 57-71, September 1983.

Since smokers experience a particularly high incidence of lung cancer, physicians are advised to search for pulmonary nodules, pleural effusions, unilateral wheezing, and persistent or recurrent pulmonary infections. When chest X-rays are insufficient, assessment by tomography or bronchoscopy is indicated. Physicians are reminded that smokers also have an increased incidence of carcinomas of the larynx, hypopharynx, tongue, esophagus, pancreas, and bladder.

84–0098. Hallgrimsson, J.; Thorarinsson, H.; Tulinius, H. **Tumours in Iceland.** *Acta Pathologica, Microbiologica et Immunologica Scandinavica; Section A: Pathology* 91A(3): 203-207, May 1983.

There is a close correlation between the sale of cigarettes and the incidence of lung carcinoma in Iceland. Among lung cancer patients with known smoking histories, all with small cell carcinoma, almost all with squamous cell carcinoma, and four out of five with adenocarcinoma were smokers. The 20-year analysis included 355 tumors (217 from males and 138 from females), and 78 percent of all registered malignant tumors of the lung. Among males, the incidence of squamous cell carcinoma and small cell carcinoma was about equal, followed by adenocarcinoma. Among females, incidence of adenocarcinoma was the highest and that of small cell carcinoma next highest; other types were much less frequent. During the period (1955 through 1974) there was a rise in the incidence of all the major histological types in both sexes, the greatest increase occurring in adenocarcinoma, doubling in males and tripling in females. It is noted that yearly per capita consumption of cigarettes rose from 200 in 1930 to 1,600 in 1979, and that approximately 64 percent of males and 45 percent of females aged 34 to 61 in Iceland are current smokers. (Auth. Abs. Mod.)

84–0099. Harris, C. C.; Trump, B. F.; Grafstrom, R.; Autrup, H. **Differences in Metabolism of Chemical Carcinogens in Cultured Human Epithelial Tissues and Cells.** *Journal of Cellular Biochemistry* 18(3): 285-294, 1982.

The metabolism of chemical carcinogens, such as those found in tobacco smoke, in cultured human bronchus, colon, duodenum, pancreatic duct, and esophagus is reviewed. The procarcinogens selected were representative of several chemical classes, and were found in drinking water, polluted air, and the diet, as well as smoke. Metabolite patterns and carcinogen—DNA adducts are generally qualitatively similar among animal species, individuals within a species, and tissues within an individual. However, wide quantitative differences are observed between individuals in outbred animal species, including humans. These interindividual differences in amounts of carcinogen-DNA adducts and in activities of enzymes that are important in the metabolism of chemical carcinogens are similar in magnitude (10- to 150-fold) to those observed in pharmacogenetic studies of drug metabolism. The role of these differences as risk factors in human cancer is being investigated. (Auth. Abs. Mod.)

84–0100. Henderson, B. E.; Kolonel, L. N.; Foster, F. H. **Cancer in Polynesians.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 62, NIH Publication No. 82-2438, December 1982, pp. 73-78.

The high incidence of lung cancer in the Polynesian populations of Hawaii and New Zealand, compared with their

European counterparts and the Polynesian population of Samoa, is associated with cigarette smoking. A 1976 New Zealand census showed that smoking was more prevalent among the Maori than among non-Maori for every age group; overall, Maori rates were 46 percent higher for males and 99 percent higher for females. However, comparison of the cigarette consumption of Polynesians and Europeans does not totally explain the elevated lung cancer rates. For example, while lung cancer incidence among Maori females, aged 40 to 49, is about four times the rate of corresponding non-Maori females in New Zealand, current smoking rates can account for only one-half this excess. No data link cigarette brands or smoking characteristics with this variation between Polynesians and Europeans, and statistics are not available concerning age of smoking onset. Despite an excess of lung cancer and cigarette smoking, a low risk of bladder cancer is seen among male Polynesians, particularly in New Zealand. General cancer patterns of the Polynesians are similar, with higher rates for stomach, breast, lung, prostate, and liver cancers, and lower rates for colon and rectal cancers. Tabulated data for incidence rates by cancer site and sex, ranking by proportional incidence, and dietary survey results are provided.

84–0101. Herity, B. **The Prevention and Early Detection of Cancer.** *Irish Journal of Medical Science* 152(6): 223-237, June 1983.

Decreasing prevalence of smoking in the Irish population suggests future reduction in lung and other smoking-related cancers. Epidemiologic data have shown smoking to be a major risk factor for lung cancer, as well as cancers of the head and neck, bladder, and pancreas. A synergistic relationship between tobacco and alcohol consumption has been demonstrated for laryngeal and tongue cancer, with particularly high laryngeal cancer risk among heavy smokers. Preventive measures must influence and accelerate changes in attitude and behavior, emphasizing health education and antismoking policies for schoolchildren aged 11 to 13. Survey statistics on numbers of smokers, ex-smokers, and nonsmokers show a declining rate for both sexes in the Republic of Ireland in 1975 and 1980 (males from 61 percent to 54 percent; females from 44 percent to 36 percent). Relative risks and synergism between tobacco and alcohol consumption are discussed and data for mortality, dietary, and sexual and reproductive behavior risk factors are provided. Also reviewed are occupational and radiation factors, and secondary prevention measures.

84–0102. Hirayama, T. **Epidemiology of Cancer of the Mouth.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute, Monograph No. 62, NIH Publication No. 82-2438, December 1982, pp. 179-183.

Case—control studies conducted in Southeast Asia show that the most important risk factor for cancers of the buccal mucosa, anterior tongue, and palate is tobacco chewing. Cigarette smoking is associated with cancer of the oropharynx, and the attributable risk for smoking and chewing combined is even higher: 88 percent for buccal mucosa, 70 percent for tongue, 28 percent for palate, and 81 percent for oropharynx. An ongoing cohort study in Japan (265,118 adults aged 40 years and older) associates combined alcohol consumption and cigarette smoking with highest risk for cancers of the mouth and pharynx (ratios 2.19 and 3.04, respectively). Data for oral and pharyngeal cancer show a dose—response relationship to total number of cigarettes ever smoked, regardless of alcohol consumption. These results contrast with esophageal and colon cancer data showing a dose—response relationship among smokers who consume alcohol daily, but not in those who consume alcohol occasionally, rarely, or never. Also provided are epidemiological data for associations between tobacco chewing/smoking and oral and pharyngeal cancer in Indian and Sri Lankan males; standardized mortality rate for mouth and pharyngeal cancer in Japanese males by cigarettes smoked/day, smoking, and alcohol consumption habits; standard mortality ratios for buccal cavity and pharyngeal cancer by number of cigarettes ever smoked and daily alcohol consumption; mouth and pharyngeal cancer mortality by socioeconomic status, sex, smoking, hot tea, and alcohol consumption; and additional mouth cancer-related rate ratios and mortality.

84–0103. Kamath, S.; Booth, P.; Lad, T. E.; Kohrs, M. B.; McGuire, W. P. **Taste Thresholds of Patients With Cancer of the Esophagus.** *Cancer* 52(2): 386-389, July 15, 1983.

Smokers with untreated esophageal cancer (n=12) had significantly higher recognition thresholds for sour, sweet, salt, and bitter tastes than nonsmoking healthy controls, but not significantly higher than matched controls who smoked. Similiar significant differences in detection thresholds were found for sour and bitter, but not for salt and sweet. Patients and 14 control subjects matched for smoking, age, and alcohol consumption, were tested for taste acuity for the four basic tastes, and evaluated by three stimulus forced choice techniques. Salivary urea nitrogen concentration was not different between patients and matched controls and did not correlate with bitter taste thresholds. Variable results have been reported for taste sensitivity in patients with malignant disease. In view of the results and the many factors that influence taste acuity, it appears that a general statement regarding taste alterations in cancer patients cannot be made. (Auth. Abs. Mod.)

84–0104. Kayser, K.; Burkhardt, H.-U. **Population Based Pathology Statistics of Oesophageal Carcinoma.** *Journal of Cancer Research and Clinical Oncology* 105(3): 292-298, 1983.

A retrospective study of esophageal cancer patients from the Regional Cancer Registry of North Baden showed a higher percentage of heavy smokers and alcohol consumers (40 percent) compared with patients with stomach, colon, and rectal cancer (10 percent). The frequency of esophageal carcinoma among autopsies performed in the Institute of Pathology in Heidelberg between 1900 and 1979 was estimated and compared with findings of the pathoanatomically based registry. The autopsy data suggest that there was no increase in the percentage of esophageal carcinomas in the area of Heidelberg during the period considered. The average age of esophageal carcinoma patients at death remained nearly unchanged during the study period, while the average age of death for all autopsies showed a steep increase. The results suggest that pathoanatomical data may be useful for checking the applicability of results obtained elsewhere. (Auth. Abs. Mod.)

84–0105. Keller, A. Z. **Selected Factors in the Risks of Upper Alimentary Cancers.** *Preventive Medicine* 12(4): 541-553, July 1983.

The failure of tobacco use to relate significantly with upper alimentary cancer (UAC) risk was unexpected, but may be due in part to the high proportion of lung cancer patients who were heavy smokers. The study investigated whether liver cirrhosis, alcohol consumption, smoking, age, occupation, and multiple primary cancers are significant risk factors for malignancies of the upper alimentary tract. It utilized maximum likelihood estimates of logistic parameters in multivariate analyses of these factors to assess risks of UAC among 529 cancer patients, 204 with liver cirrhosis (165 heavy smokers) and 325 without liver cirrhosis (212 heavy smokers). All patients were black males, discharged from U.S. Veterans Administration Hospitals from 1969 to 1975 with newly diagnosed and histologically confirmed primary cancers. In comparison with cirrhotics without UAC, cirrhotics with UAC had similar frequencies of liver cirrhosis by type, severity, and histological confirmation but were notably younger and had liver cirrhosis before cancer at a significantly higher frequency and over a significantly longer period. When simultaneous adjustment was made for all six factors, only liver cirrhosis, age, and multiple primaries (mainly at floor of mouth, soft palate, and esophageal sites) are significantly associated with increased cancer risk at upper alimentary sites. (Auth. Abs. Mod.)

84–0106. Lam, W. K.; So, S. Y.; Yu, D.Y.C. **Clinical Features of Bronchogenic Carcinoma in Hong Kong.** *Cancer* 52(2): 369-376, July 15, 1983.

Among 480 Chinese patients with proven bronchogenic carcinoma, smoking was associated with epidermoid (I) and small cell anaplastic carcinoma (II): 129 of 134 type I and 39 of 40 type II cases in males were chronic smokers, as were 35 of 50 type I and 12 of 16 type II cases in females. The relative risk of bronchial cancer in smokers was [illegible]9 (I) and 10.4 (II) in men, and 6.5 (I) and 8.3 (II) in women. Four major histologic types accounted for 87 percent of all cases: 39 percent epidermoid, 12 percent small cell anaplastic, 29 percent adenocarcinoma, and 7 percent large cell anaplastic. For all types among the 407 patients whose smoking habits were known, 244 of the 267 males, and 75 of the 140 females were smokers. The most common symptoms were anorexia, malaise, and cough. Overall, patients presented late and only 30 (6 percent) had curative surgery. The relatively rare occurrence of deep vein thrombosis (0.7 percent) is in keeping with the known low incidence of venous thrombosis in Chinese. Adenocarcinoma was a distinct group characterized by its preponderance in females (43 percent), lack of association with smoking habit (61 percent of female cases were nonsmokers), high frequency of neurologic manifestation (21 percent), and clinical, roentgenographic, and bronchoscopic features of a predominantly centrally situated tumor. Possible etiologic factors for the high and still increasing incidence of adenocarcinoma are discussed. (Auth. Abs. Mod.)

84–0107. Lee, J.; Kolonel, L. N. **Body Height and Lung Cancer Risk. (Letter).** *Lancet* 1(8329): 877, April 16, 1983.

Height was found to be a risk factor for lung cancer in both smokers and nonsmokers, and for both sexes. A study of 80 cases (37 male and 17 female smokers) and 1,081 controls (536 male and 176 female smokers) among a cohort of 50,000 adult Hawaii residents was analyzed by sex, race, smoking status, age, and socioeconomic status. Cases were noticeably (but not significantly) taller than controls, but did not differ consistently in body surface area or weight. Height exhibited a monotonic dose—response gradient in 37 male smokers (relative risk of 1, 3.2, and 3.7 for increasing tertiles of height; $\acute{p}=0.026$ for test of linear trend) and in 18 male nonsmokers (relative risk of 1, 1.5, and 2.9 for the three tertiles; $p=0.169$ for trend). The number of women (17 smokers, 8 nonsmokers) was too small for dose—response assessment by tertiles.

84–0108. Livingston, G. K.; Cannon, L. A.; Bishop, D. T.; Johnson, P.; Fineman, R. M. **Sister Chromatid Exchange: Variation by Age, Sex, Smoking, and Breast Cancer Status.** *Cancer Genetics and Cytogenetics* 9(3): 289-299, July 1983.

Data suggest that current smoking increases the sister-chromatid exchange (SCE) frequency by about 1.82 SCEs/cell, whereas sex and age have no significant effect. The mean SCE rate of smokers was consistently higher than nonsmokers in every category of comparison, supporting the validity of the smoking effect. Variation in SCE frequency in lymphocytes of 125 persons was compared to determine whether SCE frequency differs with respect to age, sex, smoking, and breast cancer status. Study subjects were divided into (1) members of two branches of families having an excess of cancer (primarily breast); (2) women in both families successfully treated for breast cancer; and (3) women from the general population with confirmed breast cancer. Controls consisted of spouses who married into the high-risk kindreds, hospital personnel, and others (primarily tradesmen without history of occupational exposure). Results show that (1) women with active breast cancer have a significantly higher mean SCE frequency than control women or women more than 5 years posttreatment for breast cancer; (2) the increase in SCE level in smokers is dose-related to exposure as measured by cumulative pack-years; (3) SCE values in both high-risk families are not significantly different from controls; and (4) the observed distribution of exchanges agrees with that expected based on the proportion of the genome represented by each chromosome group. (Auth. Abs. Mod.)

84–0109. McLaughlin, J. K.; Blot, W. J.; Mandel, J. S.; Schuman, L. M.; Mehl, E. S.; Fraumeni, J. F., Jr. **Etiology of Cancer of the Renal Pelvis.** *Journal of the National Cancer Institute* 71(2): 287-291, August 1983.

Cigarette smoking was found to be the major cause of cancer of the renal pelvis in a population-based case—control interview study of 74 cases and 697 controls conducted in the Minneapolis—St. Paul metropolitan area. The odds ratio (OR) for cigarette smoking was 7.6 among males (95 percent confidence interval (CI) 2.2 to 31.5) and 5.8 among females (95 percent CI 2.0 to 17.3), with the OR exceeding 10 for heavy smokers of both sexes. Long-term use of analgesics and several occupational exposures were also found to be risk factors. In addition, there were unexpected positive associations among females for heavy tea consumption and for use of estrogen medications, although the significance of these relationships remains unclarified. Renal pelvic cancer ORs are provided for males and females, according to pack-years of cigarette consumption. (Auth. Abs. Mod.)

84–0110. Najem, G. R.; Molteni, K. H. **Respiratory Organs Cancer Mortality in New Jersey Counties and the Relationship With Selected Demographic and Environmental Variables.** *Preventive Medicine* 12(4): 479-490, July 1983.

Because smoking and use of alcohol are more prevalent in urban, more densely populated areas, the association of respiratory cancer mortality with annual per capita income, urbanization index, and population density in males may merely reflect heavy alcohol consumption and smoking. An analysis is presented of recent rates of respiratory cancer mortality in 21 New Jersey counties during 1968-1977, a comparison with the 1950-1969 rates, and associations between current respiratory cancer mortality rates and selected demographic and environmental variables. The lung, bronchial, tracheal, and pleural cancer mortality rates among white and nonwhite males and females were substantially higher than the national rates during the period 1950-1969. Although the gaps in rates between New Jersey and the United States have narrowed, the observed number of laryngeal and lung cancer deaths remained significantly higher ($p<0.01$ to $p<0.0001$) than expected cancer deaths, among one or more subgroup populations (white and nonwhite males and females) in several New Jersey counties. Statistically significant and positive correlations were found between laryngeal cancer mortality and chemical toxic waste disposal sites (CTWDS), urbanization index, and population density. Lung cancer mortality also correlated significantly with CTWDS in New Jersey, and both larynx and lung cancer mortality showed significant and consistent negative correlations with annual per capita income. Some of the implications of the study findings are discussed and recommendations are made for future investigations. (Auth. Abs. Mod.)

84–0111. Phillips, D. H. **Fifty Years of Benzo(*a*)pyrene.** *Nature* 303: 468-472, June 9, 1983.

The isolation and identification of benzo(*a*)pyrene, through 10 years of research initiated and directed by E. L. Kennaway, is described. Following the discovery of the carcinogen, research over the next 40 years began with an investigation of its structure and biological activity. The exploration of the possibility of K-region epoxide formation, and ultimate confirmation and elucidation of metabolic activation via diol-epoxides are also reported. Recent work in assessing the human risk of benzo(*a*)pyrene (especially that present in tobacco smoke), is covered.

84–0112. Porter, J. B.; Jick, H. **Breast Cancer and Cigarette Smoking. (Letter).** *New England Journal of Medicine* 309(3): 186, July 21, 1983.

Data from two studies do not support a previous hypothesis that smoking is negatively associated with breast cancer, a theory that prompted an earlier paper (*New England Journal of Medicine* 307(17): 1062-1065, October 21, 1982) that reported a positive association between cigarette smoking and urinary estrogens. Among 5,577 patients interviewed in an intensive screening program, all women with breast cancer were matched with controls admitted for conditions considered unrelated with smoking. The second group of women were from a group health plan, also diagnosed as having breast cancer, and matched with a control group. Data on smoking habits were obtained from all groups. None of the estimated relative risks were significant in either study. The adjusted relative risk for women smoking more than a pack/day was 1.5 (surgical ward patients) and 0.92 (prepaid medical plan patients). See also 83-0188.

84–0113. Redmond, S. R. **Cancer and Tobacco Use by Sex Partners. (Letter).** *American Journal of Public Health* 73(8): 922, August 1983.

Future studies on coitus-related cervical cancer risk factors should consider the smoking habits of male partners. Cigarette smokers have been known to have mutagenic substances in their urine, and the carcinogenicity of ejaculate may be a function of tobacco consumption as well as naturally occurring substances. Thus, smoking by male partners could be an important confounding factor in epidemiological analysis of cervical cancer.

84–0114. Rilke, F. **A Place for Cytology in the Fight Against Health Consequences of Smoking.** *Pathologica* 75(1035): 144-148, January-February 1983.

Although 85 percent of lung cancer patients are moderate to heavy smokers, little interest has been shown in cytological screening for lung cancer for several reasons: the population to be screened is very large while facilities are limited; the benefits of early diagnosis are still controversial; and an in situ phase is known only for squamous cell carcinoma. The cytologic approach to diagnosis of lung cancer involves sputum cytology, examination of bronchoscopic brushing material, and fine-needle aspiration biopsy. Large-cell carcinoma and adenocarcinoma are the most frequently unidentifiable, but all cases of radiologically suspected pulmonary malignancy with negative sputum and bronchial endoscopic brushing cytology are considered eligible for fine-needle aspiration biopsy. In addition, fine-needle aspiration biopsy is an inexpensive, safe, and rapid procedure that could be performed in an outpatient clinic without major discomfort to the patient. Smoking as a socially and economically condoned habit, and Dr. Papanicolaou's role in advancing the field of cytology are also discussed.

84–0115. Rossi, L.; Barbieri, O.; Sanguineti, M.; Staccione, A.; Santi, L. F.; Santi, L. **Carcinogenic Activity of Benzo(*a*)pyrene and Some of Its Synthetic Derivatives by Direct Injection Into the Mouse Fetus.** *Carcinogenesis* 4(2): 153-156, 1983.

Results strongly support the hypothesis that 7β,8α-dihydroxy-9α,10α-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene (BPDE) is an ultimate carcinogenic metabolite of benzo(*a*)pyrene (BaP). Intrafetal injection of 0.4 nmol/fetus of BPDE was sufficient to induce lung adenomas in >50 percent of the progeny. The carcinogenic effects of direct injection of BaP and 6-methylbenzo(*a*)pyrene (BP6M) in Swiss mice were studied, as was the sensitivity of mouse fetuses to the action of ultimate carcinogens (benzo(*a*)pyrene-4,5-oxide (BPO) and BPDE). At a dose of 0.4 nmol/fetus, neither BaP nor BP6M induced tumors; injection of 4.0 or 19.8 nmol/fetus caused incidences of 53 percent and 92 percent, respectively, with BP6M, and 24 percent and 39 percent, respectively, with BaP. BPO was not tumorigenic in this system, even at doses as high as 15.7 nmol/fetus. Eleven percent of the control animals had lung adenomas. The average number of nodules/mouse varied with the compound and the dose injected, and closely followed the pattern of tumor incidence. The unique sensitivity of the injection procedure suggests its use for detecting environmental carcinogens in whole animal systems. (Auth. Abs. Mod.)

84–0116. Shimizu, H.; Preston-Martin, S.; Casagrande, J. T.; Ross, R. K.; Henderson, B. E.; Pike, M. C. **Epidemiological Characteristics of Adenocarcinoma of the Lung in Los Angeles County.** *Third Symposium on Epidemiology and Cancer Registries in the Pacific Basin.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 62, NIH Publication No. 82-2438, December 1982, pp. 161-163.

Cigarette smoking was found to be responsible for 90 percent of squamous cell carcinoma in both sexes and for 79 percent of male and 46 percent of female adenocarcinoma. The descriptive epidemiology of adenocarcinoma of the lung was compared with that of squamous cell carcinoma with the use of data for 1972 through 1976 from the population-based cancer registry serving Los Angeles County. Several differences in the demographic characteristics between these two types of lung cancer were observed, including low male-to-female ratios and lack of social class gradients for adenocarcinoma. After subtraction of the proportion of the incidence due to smoking, squamous cell carcinoma showed a fivefold excess in males compared with females, whereas rates for adenocarcinoma were almost the same in men and women. These findings indicate that (1) smoking is etiologically less important for adenocarcinoma than for squamous cell carcinoma, and (2) other etiologic factors for adenocarcinoma affect women as much as men. (Auth. Abs. Mod.)

84–0117. Sievers, M. L.; Fisher, J. R. **Cancer in North American Indians: Environment Versus Heredity.** *American Journal of Public Health* 73(5): 485-487, May 1983.

The risk of cancer is significantly lower among the Indians of North America compared with the general populations of the United States and Canada: one-half to two-thirds the national overall rate for non-Indians living in the United States, and about half the age-standardized cancer rate recorded for non-Indian populations in southwestern Canada. Cancer mortality for populations with only partial Indian ancestry are generally intermediate between the general population rates and those of tribes having full Indian heritage. Differences in smoking habits among Indian populations in the United States (full-blooded Indians in Arizona and New Mexico rarely smoke, while Oklahoma Indians with only partial Indian ancestry smoke about as much as the general population) may explain the ninefold greater lung cancer incidence in the Oklahoma Indians. While Indians appear to be susceptible to certain carcinogenic effects such as smoking, an ameliorative genetic influence may also explain the lower frequency of cancer of many sites. It may also be significant that several Indian tribes have remained in remote regions where conditions are quite different compared with the urban environments of most non-Indians. Future trends in cancer among the Indians may provide inferential evidence regarding environmental influences. Intervention to curtail expected increases in cancer rates should include innovative methods to discourage cigarette smoking and encourage diet modification.

84–0118. Simpson, S. G.; Comstock, G. W. **Lung Cancer and Housing Characteristics.** *Archives of Environmental Health* 38(4): 248-251, July-August 1983.

Only amount smoked, age, male sex, and standard of living were significantly associated with lung cancer incidence in a study of the relationship of lung cancer and indoor radon exposure in Washington County, Maryland. Lung cancer incidence was determined and correlated with personal and housing data from a nonofficial 1963 census. Because indoor radon measurements were not available, two housing characteristics reported to be related to indoor radon concentration (type of basement construction and type of building materials) were used as surrogate measures. An adjusted rate of lung cancer incidence was obtained for each characteristic. Compared with the average annual incidence of 63/100,000 population, smokers of 21 or more cigarettes/day had an adjusted rate of 141.3/100,000. Ex-smokers, cigar and pipe smokers, and those smoking less than 10 cigarettes/day had a rate of 46.2/100,000. There was no significant association between lung cancer and any of the housing characteristics, although rates were highest in houses that had concrete walls and no basements. Differences were slight, however, and could have occurred by chance. (Auth. Abs. Mod.)

84–0119. Stolley, P. D. **Lung Cancer in Women—Five Years Later, Situation Worse.** *New England Journal of Medicine* 309(7): 428-429, August 18, 1983.

Both overall smoking-related cancer mortality and increasing lung cancer mortality among women, corresponding with cigarette consumption in the 1960s and 1970s, indicate the need for a national long-range antismoking policy. Of all cancer deaths in 1982, 129,000 (30 percent) were attributed to cigarette smoking. Among women, lung cancer mortality was 4.6/100,000 in 1950, compared with 20.9/100,000 projected for 1982 and 17 percent of all cancer mortality projected for 1983. A suggested policy would include restricting cigarette smoking in public places, reducing hazardous substances in cigarette smoke, restricting cigarette advertising, disseminating information on smoking hazards, aiding persons wishing to quit, taxation and economic disincentives, and shifting of Southern agricultural emphasis from tobacco growing and cigarette manufacturing toward food production and socially useful activities. Detrimental effects of cigarette advertising include (1) inducing young persons to smoke; (2) creating a social atmosphere conducive to smoking; and (3) pressuring high-circulation publications that carry advertising not to provide antismoking and health-related information. While prevalence of smoking has recently decreased in most age groups (approximately one-third of 20- to 65-year-old women are current smokers, compared with 40 percent 18 years ago in all but the highest age groups), lung cancer rates will not reflect this trend for many years.

84–0120. Sunderman, F. W. **Cancer in Perspective. Opening Remarks at the Seminar on the Laboratory Diagnosis and Monitoring of Cancer.** *Annals of Clinical and Laboratory Science* 13(3): 169-172, May-June 1983.

It is estimated that tobacco will cause 30 percent of all cancer deaths this year, and improper diet and other lifestyle factors will be responsible for about 50 percent. The best estimates for cancer deaths from occupational and environmental causes are 4 and 2 percent, respectively. Lifestyle factors include behavioral and cultural patterns, dietary habits, nutritional factors, personal habits (e.g., cigarette smoking, alcohol consumption), carelessness about early warning signals, drug use, radiation exposure, age at pregnancy, and menopause. Most knowledge about the causes of cancer is inferential and is obtained from epidemiological data. Even though the specific etiological factors that cause lung cancer in humans (20 to 40 years after smoking onset) are not known, smokers are at increased risk of acquiring lung cancer. It is also clear from cancer epidemiology that cancer, in general, is a preventable disease.

84–0121. Tola, S. **Occupational Exposures and Lung Cancer — Epidemiological Approaches.** *European Journal of Respiratory Diseases* 63(Supplement 123): 128-138, 1982.

Smoking is related to lung cancer caused by industrial exposures for two main reasons: (1) as a major cause of lung cancer, it may act as a confounder in etiological investigations; and (2) smoking may modify the effect of other exposures, such as asbestos. Smoking acts as a confounder if it is associated with exposure in a given study. However, differences in smoking habits between occupational groups are usually not great (a range of 31 to 50 percent has been reported). Where differences are more pronounced (e.g., the construction industry, where 71 percent of painters smoke compared with only 32 percent of the civil engineers), a risk ratio variation between 0.6 and 1.4 is calculated. But such large differences are rare, and the confounding effects of smoking have probably been exaggerated. The epidemiological approaches available for recognizing and evaluating the associations between occupational exposures and lung cancer are briefly presented, with some examples. (Auth. Abs. Mod.)

84–0122. Trichopoulos, D.; Kalandidi, A.; Sparros, L. **Lung Cancer and Passive Smoking: Conclusion of Greek Study. (Letter).** *Lancet* 2(8351): 677-678, September 17, 1983.

Data are presented that increase the credibility of a hypothesis implicating passive smoking as a factor in lung cancer (*Lancet* 1(8271): 548-549, March 6, 1982). While a Greek study had been previously criticized for the small number of subjects, lack of histological confirmation, and hospital differences, there were twice the number of cases and 50 percent more controls at conclusion of the study, with results remaining substantially the same. For a total of 77 cases and 225 nonsmoking female controls, the risk ratio (RR) of lung cancer for never-married women or those with husbands who were nonsmokers or ex-smokers for 5 to 20 years (24 cases, 109 controls), was 1.0; for spouses of men who were ex-smokers for less than 5 years (15 cases, 35 controls), RR was 1.9; for spouses of men currently smoking from 1 to 20 cigarettes/day (24 cases, 56 controls), RR was 2.4; and for women whose husbands currently smoked 21 or more cigarettes/day (14 cases, 25 controls), RR was 3.4. It is stated that the small size of the RR and the many potential sources of bias preclude any single study from providing conclusive evidence; however, convergence of results from different studies in different populations will permit a sound conclusion, and the Athens study is a step in this direction. See also 82-1070, 82-1072.

84–0123. UICC Bulletin. **Thirteenth International Cancer Congress.** *UICC Bulletin* 21(1): 1-48, June 1983.

The opening address at the 13th International Cancer Congress (sponsored by the International Union Against Cancer) emphasized a continuing need to inform governments and individuals of smoking-related health hazards. While cigarette consumption has declined slightly in some Western nations, prevalence of smoking has spread rapidly in several developing countries, and is responsible for 400,000 lung cancer deaths annually. Lectures emphasized the need for nationally coordinated smoking cessation campaigns, clarification of the role of passive smoking in cancer etiology, and strategies for resolving the "tobacco problem." A special session, "Smoking and Cancer: A Comprehensive Approach," explored possibilities for changing smoking habits; current antismoking legislation; and recent advances in smoking cessation and nicotine chewing gum. Speakers agreed that change could result from citizen activity through a unified effort by voluntary societies, and that the leading cigarette producing countries (United States and United Kingdom) must take the initiative and pass health-based legislation. The most advanced antismoking programs have thus far appeared in developing countries. Nations represented at this session included Australia, United States, Austria, and Egypt, while overall participation included 4,087 presentations representing the work of over 9,000 health professionals from 84 countries.

84–0124. Vena, J. E. **Lung Cancer Incidence and Air Pollution in Erie County, New York.** *Archives of Environmental Health* 38(4): 229-236, July-August 1983.

Regression analyses did not provide any evidence for an association between ambient air trace metals or ambient benzo(*a*)pyrene (BaP) and lung cancer incidence. Total suspended particulates (TSP) comprised the strongest ambient air quality variable associated with lung cancer incidence in white males, all males, the white population, and the total population. Demographic data on race, sex, age, and address at diagnosis were obtained on the 2,201 lung cancer cases from Erie County, reported to the New York State tumor registry during the period from 1973 to 1976. Multivariate regression analysis was used to evaluate the association between age-adjusted race- and sex-specific lung cancer incidence rates by census tracts and air pollution, as indexed by TSP, several trace metals, and BaP. With the exception of white females, lung cancer incidence rates were inversely related to socioeconomic status (SES). It is suggested that high prevalence of cigarette smoking in high SES white females may be a factor. (Auth. Abs. Mod.)

84–0125. Vessey, M. P.; Baron, J.; Doll, R.; McPherson, K.; Yeates, D. **Oral Contraceptives and Breast Cancer: Final Report of an Epidemiological Study.** *British Journal of Cancer* 47(4): 455-462, April 1983.

It is suggested that data reflect the unrepresentative smoking habits of 1,176 hospital controls rather than a protective effect of smoking among 1,176 women with newly diagnosed breast cancer. Of the cancer patients, 50.7 percent were nonsmokers, 14.3 percent ex-smokers, and 35 percent smokers (over 15 percent <15 cigarettes/day). Percentages for controls were 41.3 (nonsmokers), 13.8 (ex-smokers), and 44.9 (smokers). Between 1968 and 1980, the women (aged 16 to 50 years) were interviewed about oral contraceptive use. The results were reassuring. A few statistically significant differences in oral contraceptive use were found between the breast cancer and control groups, but the data were subdivided in many ways so that some "significant" differences would have been expected through chance alone. No patterns of risk emerged which would suggest that any of the associations were causal. It is stressed, however, that data are still sparse in some important subcategories. Women who had never used oral contraceptives presented with more advanced tumors than those who had been using oral contraceptives during the year before detection of cancer, while past users were in an intermediate position. These differences in staging were reflected in the pattern of survival. Possible explanations for these observations include "surveillance bias" among oral contraceptive users leading to earlier diagnosis and a beneficial biological effect of oral contraceptives on tumor growth and spread. (Auth. Abs. Mod.)

84–0126. Watanabe, T.; Iglehart, J. D.; Bolognesi, D. P. **Secretory Immune Response in Patients with Oropharyngeal Carcinoma.** *Annals of Otology Rhinology and Laryngology* 92(3, Part 1): 295-299, May-June 1983.

The elevated immunoglobin A (IgA) levels in oropharyngeal cancer patients appear to be unrelated to a history of smoking or drinking, or to local inflammation. Amounts of secretory IgA (SIgA) and secretory component (SC) in oral secretions and sera were measured in 20 patients with oropharyngeal carcinoma and 22 controls to determine the role of the secretory immune system in oropharyngeal carcinoma. Cancer patients had increased IgA levels in oral secretions compared with normal subjects and with a smoker/drinker subgroup of controls. Density gradient analysis demonstrated that the IgA measured in oral secretions migrates at the 11S position, confirming that it is of the secretory type and indicating that the increased levels of IgA in oral secretions of patients with oropharyngeal carcinoma are not due to nonspecific leakage of 7 SIgA from sera. Secretory component levels in sera also revealed highly significant differences between cancer patients and all control subjects, suggesting that the increased SIgA concentration in secretions and SC level in sera accompany the existence of oropharyngeal carcinoma. The biological significance of these findings and their possible clinical application are discussed. (Auth. Abs. Mod.)

84–0127. Wenke, G.; Hoffmann, D. **A Study of Betel Quid Carcinogenesis. 1. On the *In Vitro* N-nitrosation of Arecoline.** *Carcinogenesis* 4(2): 169-172, 1983.

Results of in vitro experiments showed that N-nitrosation of arecoline leads to N-nitrosoguvacoline (NG), 3-(methylnitrosamino)propionitrile (MNPN), and 3-(methylnitrosamino)propionaldehyde. While NG is most likely not carcinogenic, its formation is not surprising and probably occurs by oxidative cleavage of the N-methyl bond. However, MNPN has been found to be a relatively strong animal carcinogen based on bioassays with its lower homolog. Extracts of betel quid are tumorigenic in the experimental animal, and betel quid chewing is strongly associated with cancer of the oral cavity, although no carcinogens have previously been detected in tobacco-free quid. The conditions prevailing in the oral cavity of betel quid chewers are likely to favor the formation of the three nitrosamines identified, supporting the hypothesis that during chewing, arecoline, the major alkaloid of the betel unit, gives rise to carcinogenic N-nitrosamines. (Auth. Abs. Mod.)

84–0128. Zatonski, W.; Didkowska, J.; Gadomska, H. **Cancer of the Larynx in Warsaw and in Selected Rural Areas.** *Neoplasma* 30(3): 379-384, 1983.

The dramatic increase in laryngeal cancer (LC) mortality in Poland correlates with the increase in smoking over the past 30 years. Patterns and time trends in LC incidence were analyzed for 1963 to 1977, from data in the Warsaw Cancer Registry. Laryngeal neoplasms now predominate in younger men, while esophageal cancer remains more frequent in older males. General consumption of cigarettes began after World War II, and in 1978, 70 percent of the males and 30 percent of the females were smokers (per capita consumption reached 2,613 cigarettes/year, the highest in the world). Among males aged 18 to 60, smoking rates are uniformly distributed. Among females, however, 40 percent of 18- to 24-year-olds are smokers compared with only 19 percent of those aged 40 to 59, and 7 percent of those aged 60 and over. Smoking rates also correlate with residence: 41 percent in Warsaw, 30 percent in towns, and 15 percent in the rural population. LC incidence is analyzed specifically with respect to age, sex, urban or rural residence, and other cancer sites, and data are compared with those of other countries.

See also, **84–0152, 84–0156**

NON—NEOPLASTIC RESPIRATORY DISEASES

84–0129. Abboud, R. T.; Johnson, A. J.; Richter, A. M.; Elwood, R. K. **Comparison of *In Vitro* Neutrophil Elastase Release in Nonsmokers and Smokers.** *American Review of Respiratory Disease* 128(3): 507-510, September 1983.

Chronic smoking did not affect neutrophil elastase release in vitro, and, among smokers, there was no significant relationship between in vitro neutrophil elastase release and abnormalities in lung function. Data suggest that inherent differences in elastase release from stimulated neutrophils are not likely to be of pathogenetic importance, leading to abnormal lung function in smokers. To assess whether differences in enzyme release from stimulated neutrophils could be a factor leading to increased elastase release in the lung, the in vitro release of elastase and a lysosomal enzyme, β-glucosaminidase, from neutrophils of 24 nonsmokers (mean age 39.5 years) and 30 smokers (mean age 45.4 years) were compared. Neutrophils were stimulated in vitro by zymosan as a phagocytic stimulus, and with the synthetic polypeptide, *N*-formyl-methionyl-leucyl-phenyl-alanine (FMLP), as a chemotactic factor. Smokers with abnormal lung function ($n = 13$) were older (59.8 ± 11.8 years) and had smoked more (45.6 ± 18.2 pack-years) than the 17 smokers with normal lung function (mean age, 39.8 ± 11.0 years; smoking, 22.1 ± 19.4 pack-years). There were no differences in either β-glucosaminidase or elastase release after either zymosan or FMLP stimulation among the three groups of subjects and no significant differences in the content of these enzymes in whole cell neutrophil lysates. (Auth. Abs. Mod.)

84–0130. Adams, L.; Lee, C.; Rawbone, R.; Guz, A. **Patterns of Smoking: Measurement and Variability in Asymptomatic Smokers.** *Clinical Science* 65(4): 383-392, October 1983.

Measurements of patterns of puffing (cigarette-holder pneumotachograph) and ventilation (plethysmography) were made on four separate occasions in 10 asymptomatic smokers during the smoking of a cigarette. There were marked individual differences that were consistent over 3 to 5 weeks. In itself, the pattern of smoking could be responsible for a threefold variation in smoke intake. Puffing, but not inhalation, became less intense as a cigarette was smoked. It was not possible to predict indices of absorption from smoking patterns. Certain smoking patterns (i.e., small puff volume, low puff frequency, short duration of inhalation, and expulsion of volume between puff and inhalation), may be less harmful than other characteristics and may explain why some individuals remain healthy despite a lifetime of smoking. (Auth. Abs.)

84–0131. Bresnitz, E. A.; Strom, B. L. **Epidemiology of Sarcoidosis.** *Epidemiologic Reviews* 5: 124-156, 1983.

Data suggest that smoking may have a protective effect for pathogenesis of sarcoidosis. Results of various case—control studies have shown (1) a highly significant negative association, among whites, between sarcoidosis and cigarette smoking; (2) that cases were less likely to be smokers than control patients with tuberculosis, matched for age, sex, race, and residence; and (3) a similar negative association between smoking and hypersensitivity pneumonitis in farmers, as in a recent study that demonstrated a lower prevalence in farmers who were smokers compared with never smokers. The prevalence of serum antibodies to the thermophilic organisms among asymptomatic farmers was eight times higher among those who never smoked, and six times higher among ex-smokers than among current smokers. Since (1) cigarette smoke recruits macrophages to the lung and decreases the proportion of lymphocytes, and (2) there is a decreased proliferative response of smokers' lung T-lymphocytes on exposure to various mitogens (compared with control subjects), it is suggested that sufficient interaction with the exogenous agent for development of a sarcoid-like reaction is prevented. Overall data show the highest sarcoidosis incidence rates in young adults, blacks in the United States, residents of the South Atlantic and South Central States, and residents of rural areas. Familial aggregations and lack of epidemics have also been established, while factors less definitively associated include preponderance among females and relationship to forests, farm animals, well springs, and wood fuel.

84–0132. Carel, R. S.; Greenstein, A.; Ellender, E.; Melamed, Y.; Kerem, D. **Factors Affecting Ventilatory Lung Function in Young Navy Selectees.** *American Review of Respiratory Disease* 128(2): 249-252, August 1983.

Among 528 Navy selectees 16 to 23 years of age, smoking showed a correlation coefficient of less than 0.2 to lung function measurements. Analysis of the pulmonary function data indicated that height and chest circumference were the best predictors of forced vital capacity (FVC) and forced expiratory volume in 1 sec. Only 58 (11 percent) of the subjects were designated as smokers ($>$10 cigarettes/day), and most had smoked for less than 2 years. Smoking frequency increased with age. Analysis of variance showed that the apparently greater FVC found in smokers compared with nonsmokers was related to the smokers being older (0.9 years) and taller (1.9 cm) than the nonsmokers. After adjusting for age (even in this narrow age range) and height, no difference in pulmonary function between the two groups could be demonstrated. Other factors, such as amount of sports activity, ethnicity, weight, or Quetelet index, were of minimal predictive value. (Auth. Abs. Mod.)

84–0133. Davies, S. F.; Offord, K. P.; Brown, M. G.; Campe, H.; Niewoehner, D. **Urine Desmosine Is Unrelated to Cigarette Smoking or to Spirometric Function.** *American Review of Respiratory Disease* 128(3): 473-475, September 1983.

Measurement of urine desmosine may not be useful as an indirect measurement of elastolysis in cigarette smokers. Desmosine excretion (desmosine/creatinine ratio), measured by radioimmunoassay in 157 subjects (52 nonsmokers, 46 smokers, 59 ex-smokers) did not correlate with ventilatory function (assessed by spirometry), current smoking status, or total lifetime cigarette consumption. A relationship with total pack-years was interpreted as a reflection of age and not an independent effect. Separate analysis of the few subjects with spirometric abnormalities showed that urine desmosine/creatinine was the same as that of subjects with normal spirometry. (Auth. Abs. Mod.)

84–0134. Dechoux, J.; Pivoteau, C.; Wantz, J. M. **Troubles Fonctionnels Respiratoires des Mineurs des Houilleres du Bassin de Lorraine a L'heure de la Retraite. [Respiratory Functional Changes in Lorraine Coal-Workers at the Age of Retirement.]** *Clinical Respiratory Physiology* 19(4): 385-391, 1983, French.

Among coal workers without pneumoconiosis, 20 percent of nonsmokers and 35 percent of smokers displayed ventilatory obstructive disturbances. In miners with simple pneumoconiosis, the percentage rose to 29 in nonsmokers and 52.5 in smokers. A clinical, radiological, and spirographic examination, and a measurement of lung transfer for carbon monoxide (Tco), following the steady state method, were performed in 655 coal miners. Subjects (mean age 50 years) had worked for an average of 30 years in the Lorraine coal mines. Abnormally low values of Tco were observed in 6 percent of nonsmokers and in 22 percent of smokers without pneumoconiosis, but 35 and 41 percent, respectively, in pneumoconiotic workers. Finally, by using spirography and Tco simultaneously, respiratory troubles were found in at least 63 percent of pneumoconiotic miners and in 43 percent of those without such disturbances. (Auth. Abs. Mod.)

84–0135. Do Pico, G. A.; Reddan, W.; Anderson, S.; Flaherty, D.; Smalley, E. **Acute Effects of Grain Dust Exposure During a Work Shift.** *American Review of Respiratory Disease* 128(3): 399-404, September 1983.

The prevalence of symptoms by smoking category, although greater in grain workers than in controls, was significantly different ($p < 0.05$) for sputum only in smokers and ex-smokers, and for cough, sputum, and nasal symptoms in nonsmokers. Among grain workers (118 smokers, 76 ex-smokers, 54 never smokers), wheezing and/or chest tightness were more common ($p < 0.05$) in smokers than in nonsmokers. In control subjects (87 smokers, 53 ex-smokers, 52 never smokers), cough, sputum, and nasal symptoms were significantly greater among smokers than nonsmokers. Multiple regression analysis showed that smoking did not significantly affect decreases in lung function. When lung function changes were examined by smoking category, the only significant difference observed was a smaller mean increase in maximal instantaneous expiratory flows at 75 percent of vital capacity in control subjects who were smokers, compared with those who were nonsmokers. It is concluded that occupational exposure to grain dust during a work shift has a dose-related adverse acute respiratory effect regardless of smoking habit, atopic status, or age, and produces a dose-related leukocyte response at total dust levels below 15 mg/m^3.

84–0136. Faling, L. J.; Kauffmann, F.; Kleisbauer, J.-P.; Cambon-de Mouzon, A.; Mercier, P.; Constans, J.; Blanc, M.; Rouch, Y.; Feingold, N. **Genetically Related Chronic Air-Flow Limitation or Dysanapsis With Normal Lungs? (Letters).** *American Review of Respiratory Disease* 128(2): 331-332, August 1983.

An earlier premise (*American Review of Respiratory Disease* 127(3): 263-269, March 1983) stated that the lower mean value for forced expiratory volume in 1 sec (FEV_1) in never smokers (NS-L), compared with a group of heavy smokers with high FEV_1 values (HS-H), indicated that the NS-L group suffered from chronic airflow limitation. However, it is suggested that the NS-L group may reflect airway-parenchymal dysanapsis with essentially normal lungs, and therefore the data are not relevant to the question of genetic factors in obstructive airways disease. The authors respond that the spirometric measurements do not substantiate the hypothesis of dysanaptic growth, and provide data to show that the discrepancy between FEV_1/forced vital capacity in both studies seems to be caused by the phenomenon of regression toward the mean. However, it is agreed that some of the subjects may have presented dysanaptic growth, which might be considered a genetic risk factor. It is concluded that the prevalence of symptoms reflects the importance of smoking, with differences reaching significance for morning cough and wheezing. No particular pattern of sex difference was observed, and among those with high FEV_1, symptoms may reflect a lung defense mechanism. Familial history of respiratory diseases was consistent with data on active and passive smoking. See also 83-1177.

84–0137. Florey, C. du V.; Leeder, S. R. **Methods for Cohort Studies of Chronic Airflow Limitation.** World Health Organization, Regional Office for Europe, Copenhagen, WHO Regional Publications, European Series No. 12, 1982, 140 pp.

In the epidemiological investigation of chronic airflow limitation, more experience has probably been gained through studies on the effects of air pollution, including tobacco smoke, than any other risk factor. The book was written to assist research workers in the design and execution of studies of chronic airflow limitation. All the methods described, which have been tested in the field and are known to work in principle, will need modification and adaptation to local situations. The material is organized into five chapters: (1) principles of epidemiological studies; (2) populations and sample size for cohort studies of respiratory illness; (3) examination techniques; (4) data preparation and statistical analyses; and (5) documentation. Six appendixes are included: (1) measures of reliability and validity; (2) MRC questionnaire and instructions; (3) ATS-DLD-78 questionnaire and instructions; (4) WHO children's questionnaire and notes; (5) European communities children's questionnaire and instructions; and (6) measurement of biochemical reactivity.

84–0138. Gamble, J. F.; Jones, W. G. **Respiratory Effects of Diesel Exhaust in Salt Miners.** *American Review of Respiratory Disease* 128(3): 389-394, September 1983.

Data for 258 male salt mine workers (50 nonsmokers, 58 ex-smokers, 150 smokers) confirm the detrimental effects of smoking on pulmonary function. Pack-years of smoking were significantly associated with increased cough and phlegm (but not with dyspnea), and with reductions in air and helium—oxygen spirometry (not significant for forced vital capacity (FVC)). While the age- and smoking-adjusted prevalence of cough, phlegm, and dyspnea was significantly higher than in a comparison population of blue-collar workers not exposed to lung irritants, there was no consistent association between smoking and respiratory symptoms. Comparisons within the study population showed a statistically significant dose-related association of phlegm and diesel exposure, a nonsignificant trend for cough and dyspnea, and no association with spirometry. Age- and smoking-adjusted rates of cough, phlegm, and dyspnea were 145, 159, and 93 percent compared with those found in the comparison population. Percent predicted flow rates showed statistically significant reductions, but the reductions were small and there were no dose—response relationships. Forced expiratory volume in 1 sec and FVC were about 96 percent of predicted. (Auth. Abs. Mod.)

84–0139. Garg, A.; Gopinath, P. G.; Pande, J. N.; Guleria, J. S. **Role of Radio-Aerosol and Perfusion Lung Imaging in Early Detection of Chronic Obstructive Lung Disease.** *European Journal of Nuclear Medicine* 8(4): 167-171, April 1983.

The efficacy of radio-aerosol and perfusion lung imaging in the early detection of chronic obstructive lung disease was evaluated in 38 subjects (5 nonsmokers, 21 smokers with minimal or no respiratory symptoms, and 12 patients with chronic obstructive lung disease). Each subject consented to a respiratory questionnaire, detailed physical examination, chest X-ray examinations, detailed pulmonary function tests, and ^{99m}Tc-radio-aerosol-inhalation lung imaging. Perfusion lung imaging with ^{99m}Tc-labeled macroaggregated albumin was performed in 22 subjects. A significant correlation ($p<0.001$) was observed between the degree of abnormality on radio-aerosol imaging and pulmonary function tests (PFTs), including forced expiratory volume in 1 sec, maximum midexpiratory flow rate, and mean transit time analysis. Abnormal radio-aerosol patterns and deranged PFTs were observed in 21 subjects each. Of 21 subjects with abnormal radio-aerosol pattern, 8 had normal PFTs, and 8 of 21 subjects with abnormal PFTs had normal aerosol images. Aerosol lung images and PFTs were abnormal more frequently than perfusion lung images. The results suggest that radio-aerosol lung imaging is as sensitive an indicator as PFTs for early detection of chronic obstructive lung disease, and can be usefully combined with PFTs for early detection of alteration in pulmonary physiology in smokers. (Auth. Abs.)

84–0140. Getz, S. B., Jr.; Beasley, W. E., III. **Spontaneous Pneumothorax.** *American Journal of Surgery* 145: 823-827, June 1983.

Of 400 patients admitted for treatment of spontaneous pneumothorax (317 males and 83 females, aged 0 to 60 years), 266 patients (66 percent) had a history of smoking. While the patient population was predominantly comprised of young male patients, the male to female ratio (3.8 to 1) was lower than ratios previously reported (5 to 1, or 8 to 1), possibly reflecting increasing prevalence of smoking among women. Additional factors contributing to spontaneous pneumothorax, presenting symptoms, and treatment procedures and indications are described.

84–0141. Goldhaber, S. Z.; Savage, D. D.; Garrison, R. J.; Castelli, W. P.; Kannel, W. B.; McNamara, P. M.; Gherardi, G.; Feinleib, M. **Risk Factors for Pulmonary Embolism. The Framingham Study.** *American Journal of Medicine* 74: 1023-1028, June 1983.

Smoking as a risk factor showed a borderline significance ($p=0.051$) in men but not in women among 46 subjects with major pulmonary embolism at autopsy. Potential long-term risk factors for major pulmonary embolism were assessed in 27 men and 19 women from the Framingham Heart Study with autopsy-confirmed and clinically significant pulmonary embolism. Age, systolic blood pressure, cholesterol level, cigarette use, glucose level, Metropolitan relative weight, and varicose veins had been ascertained at entry into the Study. These variables were compared among these 46 subjects, all 3,470 subjects in whom these variables were measured at the inception of the Study, and the 998 of these subjects who died within 26 years of followup. In multivariate analysis of subjects with autopsy-confirmed major pulmonary embolism and all subjects who died, only Metropolitan relative weight was significantly and independently associated with pulmonary embolism and only among women ($p<0.001$). These findings indicate that, in this cohort, increased adiposity in women is an important long-term factor for significant pulmonary embolism at autopsy, raising the possibility that weight reduction in obese women may decrease the chances of pulmonary embolism. (Auth. Abs. Mod.)

84–0142. Groth, S.; Simonsson, B. G.; Kabiraj, M.; Bulow, K. **Significance of the Relative Reduction of FVC and FEV_1 After Methacholine Challenge.** *European Journal of Respiratory Diseases* 64(Supplement 128, Part II): 409-412, 1983.

While the forced expiratory volume in 1 sec (FEV_1) was significantly reduced in smokers ($n=23$), ex-smokers ($n=23$), and nonsmokers ($n=19$) (all aged 48 to 50) after inhaled methacholine (MCL) provocation, the FEV_1 reduction in smokers was more significant. The number of FEV_1 reactors (defined according to a formula provided) at the different MCL concentrations did not differ significantly from the forced vital capacity (FVC) reactors. Three MCL percentages were inhaled for 4 minutes each, and spirometric readings were taken after each inhalation period. FVC reactors were all simultaneously FEV_1 reactors. Since FEV_1 and FVC reduction did not differ significantly among the three groups, regardless of smoking habit, the slightly reduced FEV percentage (formula provided) of the different groups was not significant. However, the FEV percentage reduction of the smokers or ex-smokers who were FEV_1 reactors was significantly higher ($p<0.02$) than that of the smoking or ex-smoking nonreactors.

84–0143. Hedenstierna, G.; Alexandersson, R.; Wimander, K.; Rosen, G. **Exposure to Terpenes: Effects on Pulmonary Function.** *International Archives of Occupational and Environmental Health* 51(3): 191-198, March 1983.

Spirometric data showed only a small (half a standard deviation) effect of terpenes, and an additive effect of smoking, on lung function. Respiratory symptoms and function were explored in 48 (24 smokers, 24 nonsmokers) terpene-exposed and 46 (16 smokers, 30 nonsmokers) nonexposed sawmill workers. A reduced forced expiratory volume in 1 sec (FEV_1) on spirometry, and an increased percentage of closing volume and slope of the alveolar plateau (phase III) on single breath nitrogen washout were seen on Monday morning before exposure to terpenes. There was no correlation between exposure time (duration of employment) and lung function impairment. Smokers showed significant reductions in all the spirometric data, regardless of terpene exposure. It is concluded that possible terpene-related obstruction does not involve any major tissues, and that cigarette smoke and terpene show only additive effects on lung function. (Auth. Abs. Mod.)

84–0144. Hogg, J. C. **Mediator Release From the Airway Mucosa.** *European Journal of Respiratory Diseases* 64(Supplement 128, Part I): 3-6, 1983.

Cigarette smoke increases the permeability of the bronchial epithelium. The resultant inflammatory reaction may be due to acquired hyperreactivity. Analysis of the inflammatory reaction produced in the airways by cigarette smoke exposure shows that epithelial permeability increases to a peak level 30 minutes after exposure and returns to normal within 24 hours. Additional data show that airways reactivity to histamine increases 30 minutes after smoke exposure and falls back toward the base level at 2 hours. (However, to obtain these results, autonomic control of smooth muscle was blocked with atropine and propranolol.) Since cigarette smoke insults to the mucosa appear to be rapidly reversible, it is stated that there should be considerable variability in the amount of mediator release by a specific challenge in the same individual. Findings suggest that mediator release from cells in the epithelium involves interactions between mediators released on the surface, the structures that maintain epithelial permeability, and the underlying nerves acting as receptors for many reflex arcs.

84–0145. Jain, S. K.; Singh, V.; Sharma, D. A. **Ventilatory Function in Chronic Bronchitis: The Value of Peak Expiratory Flow Rate.** *Indian Practitioner* 36(2): 75-82, February 1983.

Ventilatory capacity tests in chronic bronchitis patients (26 males and 16 females) showed significantly reduced peak expiratory flow rates (PEFR) and maximum ventilatory ventilation (MVV) for male smokers ($n = 16$), compared with nonsmokers. PEFR, MVV, forced expiratory volume in 1 sec (FEV_1), vital capacity (VC), and FEV_1/VC ratio were measured. Observed values for all test parameters for chronic bronchitis patients were significantly reduced when compared with respective predicted values for both sexes.

84–0146. Janoff, A. **Biochemical Links Between Cigarette Smoking and Pulmonary Emphysema.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 55(2): 285-293, August 1983.

Although the elastase—antielastase imbalance hypothesis has not been proven, recent data link this hypothesis to cigarette smoking, the primary environmental risk factor for pulmonary emphysema. It is suggested that three major chemical and cellular effects produced by cigarette smoke may be factors contributing to altered elastin metabolism and emphysema development in smokers: (1) increased recruitment of elastase—vector cells; (2) inactivation of α_1-proteinase inhibitor (α_1PI) and bronchial mucous proteinase inhibitor by cigarette smoke (perhaps via oxidation of their active-site methionine residues); and (3) interference with cross-link synthesis during elastogenesis that would otherwise occur after proteolytically mediated injury. It is suggested that all three factors would have to be present in a single individual for the final lesion to occur. Circumstantial evidence suggesting that alveolar effacement in pulmonary emphysema is due to unrestrained elastolytic activity in lung connective tissue includes (1) the proven association between genetic deficiency in α_1PI and increased incidence of the disease; (2) experimental production of emphysema through intrapulmonary instillation of elastolytic proteases; and (3) elevated elastin-degradation peptides in serum and urine of some emphysema patients. Methods of demonstrating positive correlations between cigarette smoking effects and destructive lung disease are described.

84–0147. Jones, J. R.; Higgins, I.T.T.; Higgins, M. W.; Keller, J. B. **Effects of Cooking Fuels on Lung Function in Nonsmoking Women.** *Archives of Environmental Health* 38(4): 219-222, July-August 1983.

No significant effect on forced expiratory volume in 1 sec (FEV_1) was found among nonsmoking women who reported exposure to smokers in the home, although earlier studies have shown increased incidence of lung cancer or decreased lung function in nonsmokers exposed to smoke of family members or coworkers. The case—control study of 205 women aged 20 to 39 from the Tecumseh Community Health Study compared use of cooking fuels and other factors in women from the highest and lowest quartiles of the lung function distribution. The FEV_1 was used as the index of ventilatory lung function. Data also showed that the use of a kitchen exhaust fan was significantly associated with low lung function. A larger proportion of women with low FEV_1 used gas for cooking, but this difference was not statistically significant. (Auth. Abs. Mod.)

84–0148. Kossove, D. **Smoke-Filled Rooms and Lower Respiratory Disease in Infants.** *South African Medical Journal* 61(17): 622-624, April 24, 1982.

Of 132 Zulu infants with severe lower respiratory tract disease (wheezing, bronchitis, or pneumonia (WBP) group), 15 (11 percent) were exposed to cigarette smoke, compared with only 1 of 18 with nonrespiratory problems (NRP). Of WBP infants, 93 (70 percent) had a history of daily heavy exposure to smoke from cooking and/or heating fires, compared with only 6 (33 percent) of NRP infants (highly significant, $p < 0.005$). Data for WBP relationship with breast feeding, symptoms of parents and siblings, social class, family history, and smoke exposure are provided. While a dose—response relationship is shown between parental cigarette smoking and the incidence of bronchitis and pneumonia in children under 1 year of age, it is suggested that smoke from bonfires and other fires of vegetable origin may contain higher concentrations of pulmonary irritants and carcinogens than cigarette smoke.

84–0149. Kulachkovsky, Y. V.; Kisiy, N. F.; Sachkova, A. A.; Smirnova, N. A. **Vneshneye Dykhaniye u Bol'nykh Khronicheskimi Zabolevaniyami Legkikh S Sistemnoy Arterial'noy Gipertenziey. [Smoking and Bronchopulmonary Pathology.]** *Vrachebnoe Delo* 1: 95-97, 1983, Russian.

Questionnaire results from 3,582 students showed that 54.4 percent of males and 10 percent of females smoked tobacco. While 28 percent of males and 64 percent of females began smoking during the 1st to 6th years at the Lvov Medical Institute, 76 percent of males and 68 percent of females expressed a desire to quit. Among the heavy smokers, 12 percent showed signs of chronic bronchitis. It was established that smoking affects patency of the bronchi of small and medium caliber and promotes development of obstructive ventilatory insufficiency. (Auth. Abs. Mod.)

84–0150. Leigh, J.; Outhred, K. G.; McKenzie, H. I.; Glick, M.; Wiles, A. N. **Quantified Pathology of Emphysema, Pneumoconiosis, and Chronic Bronchitis in Coal Workers.** *British Journal of Industrial Medicine* 40(3): 258-263, August 1983.

Results of postmortem examination of 886 Australian coal miners (smoking history available for 606) showed that neither postmortem emphysema nor postmortem chronic bronchitis were correlated with smoking history. Linear regression coefficients of X-ray pneumoconiosis severity (radiographs within 5 years of death) were highest in nonsmokers and correlated negatively with the amount smoked. Pneumoconiosis was significantly related to years at coal face, and face work was most prevalent in the nonsmoking group. Face work was lowest in the indeterminate smoking group with heavy smokers in the middle group. Since coal workers cannot smoke underground, at least not at the face, few smokers work at the face and are therefore exposed to less coal dust than nonsmokers. Furthermore, lighter smokers live longer, and thereby have a greater period of time for dust accumulation and pneumoconiosis development. While smoking was not correlated with severity of emphysema or bronchial gland wall ratio, there was a correlation between chronic bronchitis and smoking, suggesting that increased sputum secretion may prevent alveolar deposition of particles. Data showed a weak correlation between smoking history and age of death. Overall results reinforce the hypothesis that emphysema in coal workers is primarily related to coal dust.

84–0151. Lung Function in Epidemiology Working Group. **Comparison of "Sensitive" and Conventional Lung Function Tests.** *Clinical Respiratory Physiology* 19(4): 77p-84p, 1983.

Research discussed at a meeting on lung function testing issues in Sonderborg, Denmark, in April 1983 included (1) a cross-sectional study of lung function in tobacco workers related to cigarette smoking habits; (2) diagnostic selectivity of various lung function tests with respect to chest X-ray findings in pulmonary emphysema; (3) maximum expiratory flow-volume curves in epidemiology; (4) limitation in existing predictive equations for flow rates at low lung volumes as used in epidemiological studies; (5) definitions of sensitive tests; (6) discriminatory power of combinations of pulmonary function tests in early lung disease; (7) slope of phase III vs. classical tests and the results of an epidemiological study; (8) "conventional" vs. "sensitive" pulmonary function tests in an epidemiological study in Lorraine, France; (9) the relationship between bronchial reactivity to inhaled histamine and annual decline in forced expiratory volume in 1 sec (FEV_1) in male smokers and ex-smokers; (10) routine lung function and expiratory oxygen and carbon dioxide curves with respect to longitudinal studies in 458 coal workers; (11) multivariate growth curve analysis of data from longitudinal studies; (12) a nonlinear multivariate analysis of size and shape of maximum expiratory flow-volume curves; and (13) longitudinal vs. cross-sectional studies in measuring effects of smoking, air pollution, and hyperreactivity on vital capacity and FEV_1.

84–0152. Matthys, H.; Vastag, E.; Koehler, D.; Daikeler, G.; Fischer, J. **Mucociliary Clearance in Patients With Chronic Bronchitis and Bronchial Carcinoma.** *Respiration* 44(5): 329-337, September-October 1983.

Mucociliary clearance rates and lung function were measured in 40 normal subjects, 30 smokers with chronic bronchitis, and 64 patients with bronchial carcinoma. The mucociliary clearance rate of cancer patients was significantly lower than that of normal subjects and of patients with chronic bronchitis who smoked the same number of cigarettes. The fast clearing phase of the central bronchial tree was more delayed than the peripheral mucociliary clearance rate in smokers with and without carcinoma. The self-cleaning filter function of about the first 10 generations of the branching bronchial tree, where most bronchial carcinomas develop, was also assessed. The correlation between lung function data and smoking habits was much better ($r=0.73$) than with the measured mucociliary clearance rates ($r=0.3$). Findings suggest that inborn errors of mucociliary transport are nearly as important as external lung damage due to smoking and infection in the development of chronic bronchitis and bronchial carcinoma. (Auth. Abs. Mod.)

84–0153. Matulionis, D. H. **Pulmonary Tissue and Cigarette Smoke. 2. Parenchymal Response.** *Environmental Research* 31(1): 176-188, June 1983.

Cigarette smoke and hydrocortisone acetate (HCA) induced marked abnormalities in lungs of C57BL/6J male mice. In many pulmonary regions of smoke-exposed, HCA-treated animals, alveoli were highly congested with surfactant and flocculent material. In addition, prominent alveolar collapse and septal hypertrophy were common. These conditions resembled pulmonary proteinosis described in humans. Administration of HCA to sham-treated animals also produced lung abnormalities, although considerably milder in severity, while stress (resulting from sham treatment) or HCA injections of mice alone failed to induce any pulmonary tissue disorder. Data indicate that the genesis of abnormal conditions that resemble pulmonary alveolar proteinosis is potentiated by cumulative effects of different treatments (i.e., smoke, HCA, and stress), the most significant being the interaction between cigarette smoke and the steroid. (Auth. Abs.)

84–0154. Nakamura, H.; Izuchi, R.; Hagiwara, T.; Izumi, S.; Konishiike, J.; Omura, I.; Sakai, S.; Shigematsu, Y.; Akutagawa, M.; Takeno, Y. **Physical Constitution and Smoking Habits of Patients With Idiopathic Spontaneous Pneumothorax.** *Japanese Journal of Medicine* 22(1): 2-8, January 1983.

Tobacco consumption was significantly higher among 2,793 males (1,138 smokers) with idiopathic spontaneous pneumothorax compared with 1,994 normal controls (1,164 smokers) classified by Brinkman index. A significant relationship was also found for the height/weight ratio. The ratio of patients was lower than that of normal subjects, suggesting that idiopathic spontaneous pneumothorax is related to thin and tall individuals. Smokers were classified by total consumption levels (number of cigarettes/day times years of smoking) and divided into seven categories. In the highest consumption category, there were six smokers with idiopathic spontaneous pneumothorax compared with none among normal controls.

84–0155. Nemery, B.; Moavero, N. E.; Brasseur, L.; Stanescu, D. C. **Smoking, Lung Function, and Body Weight. (Letter).** *British Medical Journal* 287(6387): 289, July 23, 1983.

Responses (n = 3) to a published cross-sectional study of the relationship between smoking, lung function, and body weight (*British Medical Journal* 286(6362): 249-251, January 22, 1983) are individually addressed by the authors of the study. The group concurs that lung function is not necessarily unaffected if there is no overt airflow obstruction. Detailed lung function results, which were considered beyond the scope of the original paper, are provided, to show that forced expiratory volume in 1 sec was related to the subjects' slow vital capacity, and that values of vital capacity were similar in groups with and without airflow obstruction (4.29±0.64 l and 4.32±0.59 l, respectively). It is noted that the data show more than an inverse relationship between smoking and weight that might simply reflect a compulsion for oral satisfaction. The observations, confirmed in another response, show that only smokers with lungs adversely affected by smoking tend to lose weight. See also 84-0221.

84–0156. Peto, R.; Speizer, F. E.; Cochrane, A. L.; Moore, F.; Fletcher, C. M.; Tinker, C. M.; Higgins, I.T.T.; Gray, R. G.; Richards, S. M.; Gilliland, J.; Norman-Smith, B. **The Relevance in Adults of Air-Flow Obstruction, But Not of Mucus Hypersecretion, to Mortality From Chronic Lung Disease. Results From 20 Years of Prospective Observation.** *American Review of Respiratory Disease* 128(3): 491-500, September 1983.

Cause-specific mortality data for 2,718 British males previously assessed through lung function tests and questionnaires (295 never smokers, 387 ex-smokers, 1,181 light smokers (≤ 14 cigarettes/day), and 855 heavy smokers) showed that all 104 men who died of chronic obstructive pulmonary disease (COPD) in 20 to 25 years of followup were smokers. No COPD deaths occurred among the never smokers. None of the 12 ex-smokers who died had an initial ratio of forced expiratory volume in 1 sec to the cube of standing height (FEV_1/H^3) that was as much as 1 standard deviation (SD) above average, whereas 6 had initial FEV_1/H^3 values that were more than 1 SD below average. Those developing symptoms of breathlessness due to airflow obstruction may have decreased their tobacco intake, thereby producing a misleadingly flat dose—response relationship for COPD mortality when heavy and light smokers were compared. The risk of death from COPD was strongly correlated with the initial degree of airflow obstruction. However, among men with similar obstruction, age-specific COPD death rates were not significantly related to initial mucus hypersecretion, supporting the concept that airflow obstruction and mucus hypersecretion are largely independent disease processes. A moderate relationship existed between initial mucus hypersecretion and subsequent lung cancer mortality, but it is not known whether this was due solely to a common correlation of both conditions with the effective degree of exposure of the large bronchi to causative factors such as tobacco smoke. Statistics are provided on the relevance of smoking habits at the time of the original survey to mortality during subsequent decades. (Auth. Abs. Mod.)

84–0157. Plowman, P. N. **Pulmonary Macrophages of Human Smokers: Decreased Surface F_c Receptor Concentrations on Heavily Particle-Laden Cells.** *Human Toxicology* 1(4): 433-442, October 1982.

Examination of pulmonary macrophages (PM) in bronchopulmonary lavage revealed that smokers' PM tend to be larger cells with more particulate intracytoplasmic inclusions than those of nonsmokers. Electronmicrographs show that the majority of inclusions are electron dense and within lysosomes. Other lysosomal inclusions in smokers' PM are needle-like structures and lamellar bodies. A rosette assay that detects changes in surface F_c receptor expression was used to compare the F_c receptor expression of a PM subgroup that was heavily particle-laden with a "clean" PM subgroup. There is strong evidence for reduced surface F_c receptor expression in heavily particle-laden PM. The possible explanations of this difference are discussed. (Auth. Abs. Mod.)

84–0158. Rutland, J.; Cox, T.; Dewar, A.; Cole, P. **Screening for Ciliary Dyskinesia - A Spectrum of Defects of Motility and Structure.** *European Journal of Respiratory Diseases* 64(Supplement 127): 71-77, 1983.

Ciliary motility and ultrastructure were studied in 10 normal nonsmokers, 10 smokers of 10 to 40 cigarettes/day, 16 cystic fibrosis patients, 15 bronchiectasis patients, and 15 subjects with Kartagener's syndrome. The smokers, bronchiectasis patients, and Kartagener patients all had significantly greater numbers of cilia with abnormal microtubular structure than the nonsmokers and systic fibrosis patients. In cilia from Kartagener's syndrome subjects, beat frequency was slower, more microtubular abnormalities occurred, and fewer dynein arms were present than in the other groups. Ciliary motility and outer dynein arm number were positively correlated in the Kartagener patients. Ciliary beat frequency, ultrastructure, and dynein arm number were normal in smokers, cystic fibrosis patients, and bronchiectasis patients.

84–0159. Shephard, R. J.; Urch, B.; Silverman, F.; Corey, P.N.J. **Interaction of Ozone and Cigarette Smoke Exposure.** *Environmental Research* 31(1): 125-137, June 1983.

The decrease in lung function following ozone exposure was slower and the response less dramatic than previously seen in nonsmokers, suggesting the effect in the 32 chronic smokers studied may have been the delay of bronchial irritation by ozone. Possible interactions of ozone and cigarette smoke exposure were tested in 2-hour chamber exposures of the 26 male and 6 female subjects. Treatment conditions were air alone, air + cigarette smoking, ozone alone, and ozone + cigarette smoking. Ozone levels were increased progressively over four trials (0.37 ppm, 0.75 ppm, 0.50 ppm + intermittent exercise, and 0.75 ppm + intermittent exercise). Exercise was intended to increase ventilation by a factor of 2.5. Ozone exposure reduced the carbon monoxide intake normally seen with smoking, as judged from smaller increments of blood carboxyhemoglobin readings. Smoking increased heart rate both at rest and during exercise, but this response was not materially influenced by simultaneous ozone exposure. It is concluded that over an acute 2-hour exposure, there is no appreciable interaction between cigarette smoking and responses to ozone. (Auth. Abs. Mod.)

84–0160. Tager, I. B.; Weiss, S. T.; Munoz, A.; Rosner, B.; Speizer, F. E. **Longitudinal Study of the Effects of Maternal Smoking on Pulmonary Function in Children.** *New England Journal of Medicine* 309(12): 699-703, September 22, 1983.

Maternal cigarette smoking effects on pulmonary function were investigated in a cohort of children and adolescents observed prospectively for 7 years. A multivariate analysis revealed that after correction for previous forced expiratory volume in 1 sec (FEV$_1$), age, height, change in height, and cigarette smoking in the child or adolescent, maternal cigarette smoking significantly lowered the expected average annual increase in FEV$_1$ ($p=0.015$). It is estimated that if two children have the same initial FEV$_1$, age, height, increase in height, and personal cigarette smoking history, but the mother of one has smoked throughout the child's life and the mother of the other has not, the difference in the change in FEV$_1$ over time in the exposed child, compared with that in the unexposed child, will be approximately 28, 51, and 101 ml after 1, 2, and 5 years, respectively (a reduction of 10.7, 9.5, and 7.0 percent, respectively, in the expected increase). The father's smoking habit, either alone or interacting with the mother's, did not contribute to the prediction of change in the child's FEV$_1$. (Auth. Abs. Mod.)

84–0161. Teculescu, D. B.; Pham, Q. T.; Locuty, J.; Kuntz, C. **Episode Infectieux Respiratoire Banal et Obstruction des Voies Aeriennes Peripheriques. [Common Respiratory Tract Infection and Peripheral Airway Obstruction.]** *Clinical Respiratory Physiology* 19(4): 393-399, 1983, French.

The proportion of smokers was higher among the 17 subjects with a common respiratory tract infection (CRTI) than among the other 69 men examined. All had a negative medical history for respiratory diseases or symptoms. Pulmonary function was tested as part of an epidemiological study of men, aged 35 to 55 years. Subjects (45 smokers), who had normal chest radiographs and normal ventilatory function, were analyzed for the possible occurrence of a CRTI during the previous 2 months. The respiratory illness was mild in 14 of the 17 subjects, lasting from 2 to 8 days. It is concluded that a common cold does not induce disturbances of pulmonary function that are detectable after several weeks in asymptomatic males. However, the physiological pattern observed suggests minor involvement of the peripheral airways; the latter may be more severe in subjects at risk (exposed workers, heavy smokers, and the elderly) in whom abnormal pulmonary function results obtained shortly after a common respiratory infection should be interpreted with caution. (Auth. Abs. Mod.)

84–0162. Tobin, M. J.; Chadha, T. S.; Jenouri, G.; Birch, S. J.; Gazeroglu, H. B.; Sackner, M. A. **Breathing Patterns. 2. Diseased Subjects.** *Chest* 84(3): 286-294, September 1983.

The pattern of breathing was markedly altered in cigarette smokers who had few or no subjective complaints but often had evidence of small airways disease. The breathing pattern of 65 normal subjects, 22 asymptomatic smokers, 17 asymptomatic and 15 symptomatic asthmatic patients, and 28 patients with chronic obstructive pulmonary disease (COPD), 17 with restrictive lung disease, 7 with primary pulmonary hypertension, and 13 with chronic anxiety were measured utilizing respiratory inductive plethysmography. Respiratory rate was increased above the normal in smokers and in patients with COPD, restrictive lung disease and pulmonary hypertension, but remained normal in asthmatic patients. Inspiratory times of 1 second or less often occurred in patients with COPD, restrictive lung disease, and pulmonary hypertension. Smokers and patients with symptomatic asthma, COPD, restrictive lung disease, and pulmonary hypertension showed heightened respiratory center drive as reflected by elevated mean inspiratory flow. Fractional inspiratory time was reduced to a variable extent in smokers, symptomatic asthmatic patients, and patients with COPD, and was a weak indicator of airways obstruction. In smokers, the increased respiratory center drive may have been related to carryover effects of nicotine on peripheral chemoreceptors, although subjects did not smoke for 3 hours before testing. (Auth. Abs. Mod.)

84–0163. Weill, H.; Hughes, J. M.; Hammad, Y. Y.; Glindmeyer, H. W.; Sharon, G.; Jones, R. N. **Respiratory Health of MMMF Workers in the United States.** In: World Health Organization. *Biological Effects of Man-Made Mineral Fibres.* Report on a WHO/IARC Meeting, EURO Reports and Studies 81, Copenhagen, 1982, pp. 93-94.

Prevalence of small lung opacities was positively associated with age and smoking among 1,028 men employed an average of 18 years in mineral wool and glass fiber plants. Median level of exposure to an average concentration of fibers ≤ 3 µm was 0.064 fibers/ml. Lung function and increased incidence of respiratory symptoms were significantly associated with smoking. Prevalence of small opacities was only 11 percent, and no large opacities were found. After controlling for smoking, age, height, and race, no adverse effect on lung function was detected for man-made mineral fiber (MMMF) exposure. It is concluded that exposure to MMMF may lead to a low-level profusion of small opacities, but a diffuse tissue reaction seems unlikely.

84–0164. Zammit-Tabona, M.; Sherkin, M.; Kijek, K.; Chan, H.; Chan-Yeung, M. **Asthma Caused by Diphenylmethane**

Diisocyanate in Foundry Workers. Clinical, Bronchial Provocation, and Immunologic Studies. *American Review of Respiratory Disease* 128(2): 226-230, August 1983.

Among 11 foundry workers who developed asthma, smoking was unrelated to provocation test responses with formaldehyde and diphenylmethane diisocyanate (MDI), components of PepSet used for making molds for casting. Of the seven who reacted, three smoked and one was an ex-smoker, while two of the nonreactors smoked and one was an ex-smoker. Six subjects showed specific asthmatic reactions to MDI challenge (more than a 20 percent fall in forced expiratory volume in 1 second); two had a combined immediate and late reaction, and four had a predominantly late reaction. Four of the six subjects experienced recurrent nocturnal asthmatic reactions after a single challenge with MDI, and one subject had an immediate irritant reaction to both MDI and formaldehyde that was spontaneously reversed in a few minutes. None of the remaining 10 subjects had a positive reaction to formaldehyde challenge. The reactors to MDI showed more evidence of airflow obstruction in lung function measurements and a greater bronchial sensitivity to methacholine than the nonreactors, but there were no differences in smoking habits, atopic status, age, or immunologic findings. It is concluded that regardless of smoking habits, MDI is a cause of asthma in foundry workers. (Auth. Abs. Mod.)

See also, **84–0089, 84–0281**

CARDIOVASCULAR DISEASES

84–0165. Campeau, L.; Enjalbert, M.; Lesperance, J.; Vaislic, C.; Grondin, C. M.; Bourassa, M. G. **Atherosclerosis and Late Closure of Aortocoronary Saphenous Vein Grafts: Sequential Angiographic Studies at 2 Weeks, 1 Year, 5 to 7 Years, and 10 to 12 Years After Surgery.** *Circulation* 68(3, Part II): II-1-II-7, September 1983.

Development of atherosclerotic lesions was not related to smoking, other classic risk factors, age, or extent of coronary artery disease before aortocoronary saphenous vein bypass surgery. Of 82 unselected patients (68 males, 14 females), 22 were never smokers, 27 had stopped smoking prior to surgery, and 33 were current smokers (half had stopped at the time of surgery). Sequential control angiographic examinations were performed at 2 weeks, 1 year, 5 to 7 years, and 10 to 12 years after surgery. Graft modifications that developed after the first year were attributed to atherosclerosis. Incidence, which increased from 16 percent between 1 year and 5 to 7 years, to 36.4 percent between 7 and 12 years, was not influenced by the severity of early diffuse or localized intimal hyperplasia. Late graft closure may result from early localized stenosis related to improper surgical techniques, but the most frequent cause appears to be atherosclerosis. Graft patency at 10 to 12 years was 63.3 percent. (Auth. Abs. Mod.)

84–0166. Chiba, K.; Koizumi, A.; Kumai, M.; Watanabe, T.; Ikeda, M. **Nationwide Survey of High-Density Lipoprotein Cholesterol Among Farmers in Japan.** *Preventive Medicine* 12(4): 508-522, July 1983.

Nondrinking males who smoked showed the lowest levels of high density lipoprotein (HDL) cholesterol among 788 men and 1,346 women in Japan (2,134 blood samples collected from farmers in 44 districts). Mean serum HDL level was 47.3±14.2 mg/100 ml in men and 47.4±12.8 mg/100 ml in women. Smoking was negatively associated ($p<0.05$) with HDL in men. Alcohol consumption was associated with elevated HDL levels, but was significant only in men. HDL levels in the nonsmoking, nondrinking population were 47.0±12.8 in women ($n=900$), and 45.5±12.5 mg/100 ml in men ($n=60$). HDL values did not differ significantly by sex and were essentially the same in husbands and wives. Among 535 subjects from whom samples were taken twice, HDL was significantly higher in summer than in winter. HDL values also varied significantly by region.

84–0167. Chronic Diseases in Canada. **Oral Contraceptives, Age, Smoking and Circulatory System Disease.** *Chronic Diseases in Canada* 4(1): 7-9, June 1983.

In women who smoke heavily, the risk of heart attack is estimated to be 7 to 34 times that of nonsmokers, and for women who smoke heavily and use oral contraceptives (OCs), the risk is even higher (relative risk 7.8 for heavy smokers, 39.0 for heavy smokers using OCs). The relative risk of circulatory disease mortality is higher in women over age 35 who smoke, with oral contraceptive use an added risk factor. An excess of 16.5 deaths (per 100,000 woman-years) for women over age 45 who smoke, 40.9 for those using OCs, and 178.8 for those who smoke and use OCs has been recorded, compared with women who do not smoke or use OCs. Other specific circulatory system diseases studied indicate that OC use and smoking may be factors in increased risk of venous thromboembolic disease, ischemic heart disease, and stroke. It is concluded that women under age 35 can use the pill without added risk of death from circulatory disease, but they should not smoke. Women over age 45 should use the pill only in exceptional circumstances.

84–0168. Coffman, J. D. **Principles of Conservative Treatment of Occlusive Arterial Disease.** *Cardiovascular Clinics* 13(2): 1-13, 1983.

Treatment of intermittent claudication (IC) must include cessation of smoking, and it has been recommended that patients be refused surgery until they stop smoking. IC is twice as frequent among smokers than nonsmokers (73 to 90 percent of patients smoke), and risk increases with higher cigarette consumption. A Mayo Clinic study showed that 11 percent of the arteriosclerosis obliterans patients who smoked required amputation over a 5-year period, although none of those who quit needed amputation. In other research, 25 percent of aortofemoral grafts and 40 percent of femoropopliteal grafts occluded in smokers, compared with only 10 percent among nonsmokers. How tobacco smoke induces or aggravates arteriosclerosis obliterans is not known, but mechanisms that are potentially responsible, involving carbon monoxide and nicotine, are reported. The discussion of risk factors also includes diabetes, hypertension, and hyperlipoproteinemia. Treatments utilizing exercise, vasodilator drugs, and fibrinolytic, anticoagulant, and antiplatelet therapy, are reviewed.

84–0169. Cooke, K. M.; Frost, G. W.; Stokes, G. S. **Blood Pressure and Its Relationship to Low Levels of Alcohol Consumption.** *Clinical and Experimental Pharmacology and Physiology* 10(3): 229-233, May-June 1983.

Multiple regression analysis for both males and females showed that smokers had lower blood pressure than nonsmokers, independent of age, alcohol consumption, and adiposity. The association between average weekly alcohol consumption and blood pressure was studied in relation to smoking, age, and adiposity, in 13,535 males and 7,385 females. Analysis of the relative contribution of each independent variable to diastolic blood pressure showed that smoking was the least significant, compared with Quetelet index and alcohol consumption. Apart from the coefficients for smoking habit, all the partial regression coefficients for systolic blood pressure were statistically significant among both men and women. The smoking habit regression coefficient was significant in women only. These regression equations accounted for 12 percent of the variation in systolic blood pressure among men and 23 percent among women. While results associate a progressive increase in blood pressure with increasing alcohol consumption (including the level of 10 to 60 g of ethanol/week), this became apparent for women only after adjusting jointly for differences in age, adiposity, and smoking.

84–0170. Cullen, K.; Stenhouse, N. S.; Wearne, K. L.; Welborn, T. A. **Multiple Regression Analysis of Risk Factors for Cardiovascular Disease and Cancer Mortality in Busselton, Western Australia—13-Year Study.** *Journal of Chronic Diseases* 36(5): 371-377, 1983.

In comparing 13-year data from Busselton with results of the Framingham study, it was found that smoking was a significant risk factor in deaths from all causes in both men and women; 40- to 59-year-old women and 60- to 74-year-old men in Busselton; and in all men and women in the Framingham group. In Busselton, smoking also increased the risk of cancer in 60- to 74-year-old women, but was unrelated to cardiovascular disease (CVD) or coronary heart disease (CHD) in either men or women. Busselton subjects ($n = 1,564$) were followed from 1966 to 1977 to determine whether risk factors for CVD and CHD differed from Framingham results. In the subjects aged 40 to 59 at entry, raised CVD mortality in men was found to be related to age, systolic blood pressure (SBP), cholesterol, and negatively to forced expiratory volume in 1 sec (FEV_1). CHD in men was related to age and to cholesterol. Raised mortality due to all causes in men was related to age, systolic blood pressure (SBP), and negatively to FEV_1 and insulin. Deaths due to all causes in women were related to age, weight, and smoking. In subjects aged 60 to 74 at entry, CVD mortality in men was related to age and to SBP. CVD in women was related to age, blood glucose and negatively to FEV_1. CHD mortality in men was related to SBP. Deaths due to all causes were related in men to age, insulin, smoking, and negatively to FEV_1. Important negative findings were the absence of serum calcium as a CVD risk factor and the confirmation that symptomless hyperuricemia is not associated with increased mortality.

84–0171. Daly, L. E.; Mulcahy, R.; Graham, I. M.; Hickey, N. **Long Term Effect on Mortality of Stopping Smoking After Unstable Angina and Myocardial Infarction.** *British Medical Journal* 287(6388): 324-326, July 30, 1983.

Results suggest that cessation of smoking is the most effective single action in the management of patients with coronary heart disease. A total of 498 men (under 60 years of age), who had survived a first episode of unstable angina or myocardial infarction by 2 years, were followed up by life table methods for a further 13 years. Mortality in those who continued to smoke was significantly higher (82.1 percent) than in those who stopped smoking (36.9 percent). These differences increased with time. Mortality in those who were nonsmokers initially and who continued not to smoke was intermediate (62.1 percent). The adverse effect of continued smoking was most pronounced in those with unstable angina. Continuing to smoke increased the rate of sudden death to a greater degree in those with less severe initial attacks, while the effect of smoking on fatal reinfarctions was most apparent in those with a more complicated presentation. (Auth. Abs. Mod.)

84–0172. DeGoulet, P.; Menard, J.; Vu, H.-A.; Golmard, J.-L.; DeVries, C.; Chatellier, G.; Plouin, P.-F. **Factors Predictive of Attendance at Clinic and Blood Pressure Control in Hypertensive Patients.** *British Medical Journal* 287(6385): 88-93, July 9, 1983.

Cigarette smoking was found to be significantly associated with dropping out of a hypertension treatment program ($p < 0.001$). Factors predictive of poor attendance and inadequate blood pressure control in patients of a hypertension clinic were determined using univariate analyses and a multivariate logistic model. Of patients with blood pressure exceeding 160/95 mmHg, 209 of the 1,346 dropped out the first year. Other variables that were significantly related to increased dropout rates during 3-year followup were male sex, youth, obesity at entry, direct referral to the clinic as a result of screening rather than by a general practitioner, absence of preexisting antihypertensive treatment at the first visit, moderate hypertension, and low socioeconomic status. Variables at entry significantly related to poor blood pressure control at 1 year were old age, evidence of coronary heart disease, severe hypertension, and raised blood glucose concentrations. Characteristics acquired before clinic consultation, such as cigarette smoking, were perceived as reflecting an uncooperative attitude toward cardiovascular risk prevention. (Auth. Abs. Mod.)

84–0173. Dickerson, J. **The Pill. A Closer Look.** *American Journal of Nursing* 83(10): 1393-1398, October 1983.

Among oral contraceptive (OC) users, female smokers over 35 years of age are most likely to suffer morbidity or mortality from vascular and circulatory disorders, the risk increasing with quantity smoked. For each 100,000 OC users, from 180 to 200 of those over age 35 could die each year if they also smoke. A smoker aged 35 to 44 years has a mortality risk of 50/100,000, while only 10 smokers and 2 nonsmokers under age 35 would be so affected. The risk for myocardial infarction for a heavy smoker using OCs is five times that of a heavy smoker who does not use OCs. Female smokers over age 35 are advised to use alternate contraceptive methods. Provided are tabulated data for relative risk of heart attack associated with cigarette smoking and OC use, compared with nonsmoking nonusers; and circulatory disease mortality rates and risks by age, smoking status, and OC use.

84–0174. Durakovic, Z.; Mimica, M. **Navika Pusenja i Promjene u Elektrokardiogramu. [Smoking Habit and Changes in the Electrocardiogram.]** *Arhiv za Higijenu Rada i Toksikologiju* 33(4): 307-313, 1982, Serbo-Croation.

Smoking-related changes [illegible] the electrocardiogram were analyzed in 2,161 women and 2,049 men aged 35 to 54 years from six regions of Croatia. Data on smoking habits were collected with a questionnaire. Nonsmokers were those who had never smoked, who smoked less than one cigarette/day, or less than 25 grams of tobacco/month, for less than 1 year. Former smokers were subjects who had ceased smoking at least 1 month before examination. Present smokers were classified as having smoked for up to 20 years, or for over 20 years. In smokers, there were no significant Q-wave findings. S-T segment depression was not present in male smokers, but was significantly more frequent among female smokers than in other groups. Inverted T-wave was more often present in male smokers and former smokers and in women former smokers than in others. Left axis deviation was more often found in women smokers and former smokers. Incomplete right bundle branch block, sinus tachycardia, S-T segment elevation, and high T-wave amplitude were frequent findings in male smokers, and incomplete right bundle branch block and sinus tachycardia were common in female smokers. The necessity for prospective investigations of the relationship between smoking and ECG signs of coronary heart disease is pointed out. (Auth. Abs. Mod.)

84–0175. Fox, K. M.; Deanfield, J.; Selwyn, A.; Krikler, S.; Wright, C. **Factors Influencing the Treatment of Chronic Stable Angina Pectoris with Nifedipine.** *Postgraduate Medical Journal* 59(Supplement 2): 25-29, 1983.

Analysis of the interaction between smoking, nifedipine, propranolol, and atenolol showed that smoking directly affected the usual beneficial effects of nifedipine and propranolol, but not atenolol. Ten exertional angina patients (six males, four females) who smoked 10 cigarettes/day were studied using 16-lead precordial exercise testing. Each underwent four 1-week double-blind treatments of placebo, nifedipine 60 mg/day, propranolol 240 mg/day, and atenolol 100 mg/day, repeated on and off smoking. Heart rate was greater during the smoking treatments, and most pronounced with nifedipine. All three drugs showed an improvement over placebo in terms of work load to chest pain and S-T segment depression whether or not patients were smoking, though atenolol appeared to be the least affected by the detrimental effects of the cigarettes. Although all treatments, including placebo, showed a reduction in the exercise-induced area and severity of S-T segment depression when the patients stopped smoking, the effects of atenolol were found to be the least altered by smoking. Blood levels of nifedipine and propranolol tended to be greater when the patients stopped smoking; the blood level of atenolol was unchanged. It is concluded that smoking caused direct and adverse effects on the heart that were still evident, though less obvious, with atenolol. Part of this differential effect may be related to the known different modes of metabolism of the drugs. A separate study analyzed effects of increasing nifedipine doses in angina patients. (Auth. Abs. Mod.)

84–0176. Friedman, G. D.; Fireman, B. H.; Petitti, D. B.; Siegelaub, A. B.; Ury, H. K.; Klatsky, A. L. **Psychological Questionnaire Score, Cigarette Smoking, and Myocardial Infarction: A Continuing Enigma.** *Preventive Medicine* 12(4): 533-540, July 1983.

Responses to a 94-item psychological questionnaire designed to elucidate the susceptibility of cigarette smokers to myocardial infarct (MI) failed to yield clear-cut results. Statistically, there was considerable interaction in relation to relative risk (RR) of MI among the 222 cases and 228 controls, but in the highest quartile (23-70 of the responses) the positive association between smoking and risk of MI disappeared (0.7 RR compared with 3.8 in the first quartile, 3.2 in the second, and 1.5 in the third). The ability of the psychological questions to discriminate between men in whom cigarette smoking was associated with increased risk of MI and men in whom smoking was not so associated remains puzzling. Further analyses, controlling for reported alcohol consumption and for a questionnaire item that might reflect physical activity, failed to alter this finding. This interaction of the questionnaire responses with smoking was not found with two other major coronary risk factors, serum cholesterol and systolic blood pressure. Believing that these observations may provide (1) a clue to how cigarette smoking affects risk of MI, or (2) some means of identifying greater or lesser susceptibility to the effects of smoking, other researchers are urged to join the investigation. A list of 10 selected yes-or-no questions with strong interaction with smoking is provided to assist others in studying this phenomenon. Items are similar to 10 found on the Minnesota Multiphasic Personality Inventory. (Auth. Abs. Mod.)

84–0177. Hay, D. R.; Beaglehole, R.; Gwynne, J. F.; Leslie, P. N.; North, J.D.K.; Nye, E. R.; Sharpe, D. N.; Simpson, F. O. **Coronary Heart Disease: Prevention and Control in 1983.** *New Zealand Medical Journal* 96(731): 346-350, May 11, 1983.

An abridged version of the strategies and recommendations made by a committee of the National Heart Foundation of New Zealand for control of coronary heart disease is presented. Recommendations for control include the risk factors of smoking, serum cholesterol, blood pressure, physical activity, and body weight. Preventive strategies focus specifically on primary prevention in the overall population and in high-risk subgroups, secondary prevention, and primordial prevention during the westernization of South Pacific cultures. The goal for smoking is a reduction in the proportion of smokers from 32 to 20 percent of the population within a decade (self-reported smoking rates have been declining significantly since 1976). The committee calls for (1) prohibition of all forms of tobacco promotion, (2) an increase in cigarette prices, (3) antismoking education, (4) provision of cessation clinics, (5) encouragement of nonsmoking in public places, and (6) promotion of nonsmoking as the social norm.

84–0178. Hay, D. R.; Beaglehole, R.; Gwynne, J. F.; Leslie, P. N.; North, J.D.K.; Nye, E. R.; Sharpe, D. N.; Simpson, F. O. **Coronary Heart Disease. A Report on Prevention and Control in 1983.** Green Lane, Auckland, The National Heart Foundation of New Zealand, May 1983, 177 pp.

The first of six major recommendations by the National Heart Foundation of New Zealand, made in 1971, for the prevention of coronary heart disease (CHD) was that cigarette smoking should be discontinued (or never begun), and every effort should be made to curtail or prohibit the habit since it can have such serious consequences for those predisposed to CHD. Trends in CHD in 1980 show that it remains the major cause of death, although there has been a decline in rates since 1968. While the dose—response relationship between smoking and CHD has been shown to be independent of other risk factors, smoking also acts synergistically with some factors in CHD risk. Census information shows that professionals smoke less than sales and service workers, and women smoke less than men in all categories except professional workers. Among the occupational groups listed, doctors had the lowest percentage of smokers (20 percent of men, 17 percent of women). Overall smoking rates declined from 46 percent to 39 percent in men and from 39 percent to 29 percent in women between 1975 and 1981. It is noted that smoking filtered or low-tar cigarettes probably does not reduce the association between smoking and CHD, whereas cessation after myocardial infarction improves prognosis. Other risk factors for CHD discussed are diet, blood pressure, exercise, alcohol, oral contraceptives, diabetes mellitus, psychosocial factors, lipids, and lipoproteins. Also reviewed are pathogenesis; patient identification, care, and treatment; coronary artery surgery; and use of drugs. Actions taken on previous recommendations are listed, and strategies for the prevention of CHD are recommended.

84–0179. Henningsen, N. C.; Janzon, L.; Trell, E. **Influence of Carboxyhemoglobin, Gamma-Glutamyl-Transferase, Body Weight, and Heart Rate on Blood Pressure in Middle-Aged Men.** *Hypertension* 5(4): 560-563, July-August 1983.

Carboxyhemoglobin (COHb percent), measured as an indicator of cigarette smoke exposure, was negatively correlated to blood pressure (BP) in 242 48-year-old men attending a general health screening program in Malmo, Sweden. None of the subjects was under treatment for high BP, and all were instructed to abstain from smoking during the mornings prior to and during the study. Serum gamma-glutamyl-transferase (GGT) was assessed as reflecting alcohol consumption. Heart rate showed a positive correlation to all the BP values. COHb percent correlated negatively to diastolic BP (DBP) ($r=-0.16$, $p<0.01$). Body weight was positively related to DBP and weakly to systolic BP ($r=0.26$, $p<0.001$ and $r=0.15$, $p<0.01$, respectively). GGT was positively correlated with DBP ($r=0.22$, $p<0.01$).

84–0180. Hossack, K. F.; Bruce, R. A.; Fisher, L.; Hofer, V. **Prognostic Value of Risk Factors and Exercise Testing in Men with Atypical Chest Pain.** *International Journal of Cardiology* 3(1): 37-50, April 1983.

Regression analysis (Cox's model) confirmed a significant association of smoking ($p=0.03$), elevated systolic blood pressure ($p=0.02$), and hypercholesterolemia ($p=0.03$) with primary coronary heart disease events. Among 551 men (201 smokers) with atypical chest pain enrolled in the Seattle Heart Watch Study, annual followup over a 5- to 10-year period identified 36 persons who experienced a primary coronary heart disease event. The three fatal events were in men over the age of 50. Event rate in smokers was 15.7/1,000 man-years compared with 7.7/1,000 man-years in nonsmokers. A combination of all three risk factors was highly predictive ($p=0.002$), while a positive family history was not. Functional aerobic impairment ($p=0.002$) and S-T depression ($p=0.0003$) were the most useful exercise predictors. In men with neither risk factors nor abnormal exercise predictors, 96 percent were free of coronary events at 9 years compared with 76 percent in men with one or more of the abnormal exercise findings ($p<0.0001$). Of men with risk factors only, 86 percent remained free of events at 9 years. (Auth. Abs. Mod.)

84–0181. Jenkins, C. D.; Stanton, B.-A.; Klein, M. D.; Savageau, J. A.; Harken, D. E. **Correlates of Angina Pectoris Among Men Awaiting Coronary By-Pass Surgery.** *Psychosomatic Medicine* 45(2): 141-153, May 1983.

A history of smoking was a predictor of angina in 204 men awaiting coronary artery bypass graft surgery in an investigation of the biomedical, behavioral, and psychological correlates of angina pectoris. Angina was rated by a precoded series of interview questions, and four circumstances of symptoms were investigated: exertional, emotional, postprandial, and during rest or sleep. These were uncorrelated with one another, except for exertional and postprandial. Two-thirds of the patients experienced angina less often than daily in the most recent unrestricted month, and severity of coronary artery obstruction was not positively associated with frequency or severity of any type of angina. The stronger predictors, which varied by type of angina, were primarily behavioral and psychological. Index of lifetime smoking was the strongest correlate with exertional angina and correlated highly with resting angina, but showed less correlation with emotional and none with postprandial angina. Disturbances of sleep, physical inactivity, distressed response to life crises, life dissatisfactions, hostility, use of propranolol, duration of cardiac illness, and age were also predictors in the multiple regression equations. These results from selected bypass candidates may apply more directly to such persons than to unselected community residents reporting angina symptoms. The findings suggest the need for greater focus on sources of variability in myocardial oxygen supply and demand in understanding the dynamics of angina episodes. (Auth. Abs. Mod.)

84–0182. Johansson, S.; Vedin, A.; Wilhelmsson, C. **Myocardial Infarction in Women.** *Epidemiologic Reviews* 5: 67-95, 1983.

A review of the current state of knowledge of coronary heart disease (CHD) among women found that smoking is significantly associated with overall CHD, nonfatal primary myocardial infarction, degree of atherosclerosis, sudden CHD death, and may be weakly associated with angina pectoris. The risk increases with increasing consumption, and women who smoke heavily are a mean of 19 years younger at death than nonsmoking women. Other risk factors discussed include hypertension, serum lipids, diabetes, obesity, oral contraceptives and noncontraceptive estrogens, menopause, psychosocial factors, familial aggregation, and the effects of multiple risk factors. The incidence, prevalence, mortality, and prognosis for CHD among women are also reviewed.

84–0183. Kashani, J. H.; Frank, R. G.; Kashani, S. R.; Wonderlich, S. A.; Reid, J. C. **Depression Among Amputees.** *Journal of Clinical Psychiatry* 44(7): 256-258, July 1983.

Of 65 amputees evaluated for depression, 20 (65 percent) of those whose amputations were due to vascular disease ($n=31$) had been smoking for 30 to 40 years, while only 12 of the remaining 34 patients had ever smoked. Findings indicated a significantly higher prevalence of major depressive disorder ($n=23$, 35 percent) than would be expected in the general population, and significantly more alcohol abuse within the depressed group. More females than males were depressed; 70 percent of males were married, compared with 40 percent of females. There were no significant differences between males and females in terms of smoking, alcohol abuse, socioeconomic status, or number of amputations.

84–0184. Layde, P. M.; Ory, H. W.; Beral, V.; Kay, C. R. **Incidence of Arterial Disease Among Oral Contraceptive Users.** *Journal of the Royal College of General Practitioners* 33(247): 75-82, February 1983.

Cigarette smoking increased the risk of arterial disease for older women, but had little effect on women under 35 years of age. The greatest risk was in current oral contraceptive (OC) users over age 35 who also smoked (3.1 times greater compared with nonsmoking controls). The incidence of all initial episodes of arterial disease was investigated among 23,000 women taking OCs and an equal number of controls (never users). In general, findings were consistent with earlier reports: (1) risk of ischemic heart disease, cerebrovascular disease, and peripheral vascular disease was increased in current users of OCs; (2) smoking increased the risk of arterial disease in both pill users and controls; and (3) risks were greatest in older women. Among 223 current OC users with initial arterial disease, 127 were smokers and 96 were nonsmokers; in 140 current users over age 35, 86 were smokers and 54 were nonsmokers. The case fatality rates for users who smoked were two to three times greater than those for women in other groups, emphasizing the combined adverse effects of OC use and smoking. It is also noted that the risk of cerebrovascular disease remained elevated in former users for at least 6 years.

84–0185. Lewis, B.; Schettler, G. **Opinion Report. Vox Populi: A Survey of the Views of a Sample of the Membership of the International Atherosclerosis Society on Coronary Heart Disease Prevention. (Letter).** *Atherosclerosis* 47(1): 107-110, April 1983.

A random sampling of the membership of the International Atherosclerosis Society (69 percent response) showed that the majority of the clinicians and research workers regard cigarette smoking, high plasma cholesterol levels, hypertension, and diabetes as causes of coronary heart disease (CHD) and not merely predictive factors. There was no consensus as to whether personality factors or sedentary lifestyle are among the causes of CHD. While no field of study is without areas of controversy, this survey was an attempt to identify areas where there was near unanimity or a large measure of consensus, since opposition to preventive measures against CHD has often been the claim that medical opinion is divided as to their value. Respondents generally agreed there was adequate evidence to advise reduction in weight and dietary cholesterol, increased intake of linoleic acid, and reduction in alcohol consumption. The strongest consensus was on the need to reduce saturated fat intake and give up smoking.

84–0186. Mason, C.; Murdoch, J. C. **Current Opinion Concerning the Treatment of Heart Disease.** *Journal of the Royal College of General Practitioners* 33(247): 91-99, February 1983.

Using a postal questionnaire, the views of 155 Tayside general practitioners were examined with respect to their management of three hypothetical patients with ischemic heart disease (IHD). Advice about smoking was offered by 73.1 percent: 46.2 percent mentioned smoking to both sexes and 26.9 percent to males only. Two suggested a reduction in smoking, while the remainder recommended cessation. Advice given to patients concerning IHD risk factors tended to be general rather than specific and emphasized restriction of activities rather than return to normal life. Commonly held social stereotypes appeared to affect the advice practitioners said they would offer. The implications of the findings are discussed. (Auth. Abs. Mod.)

84–0187. Rechnitzer, P. A.; Cunningham, D. A.; Donner, A. P.; Andrew, G. M.; Buck, C. W.; Jones, N. L.; Kavanagh, T.; Oldridge, N. B.; Parker, J. O.; Shephard, R. J.; Sutton, J. R. **Characteristics That Predicted Recurrence of Infarction Within 3 Years in the Ontario Exercise-Heart Collaborative Study.** *Canadian Medical Association Journal* 128(11): 1287-1290, June 1, 1983.

Smoking and blue-collar occupation were independently and significantly ($p<0.05$) predictive of recurrence of myocardial infarction in 618 patients in the Ontario Exercise-Heart Collaborative Study. Data on 13 baseline characteristics of myocardial infarction patients were analyzed to determine their value in predicting infarction recurrence within 3 years. Of the subjects with recurrence, 56 percent were current smokers, while only 33 percent of those without recurrence were smokers. The estimated adjusted relative odds were 2.3 for smoking and 2.1 for blue-collar occupations. Other characteristics considered were age, serum levels of cholesterol and triglycerides, systolic and diastolic blood pressure, number of hours of competitive sports/week, number of hours of jogging/week, angina, cough and phlegm production, personality type, and the number of previous infarctions. (Auth. Abs. Mod.)

84–0188. Shekelle, R. B.; Gale, M.; Ostfeld, A. M.; Paul, O. **Hostility, Risk of Coronary Heart Disease, and Mortality.** *Psychosomatic Medicine* 45(2): 109-114, May 1983.

Analysis of data according to quintile of the hostility (Ho) scale showed small but statistically significant associations with number of cigarettes smoked/day, age, and alcohol consumption, but not systolic blood pressure or serum cholesterol level. Level of Ho was assessed by a 50-item subscale of the Minnesota Multiphasic Personality Inventory at the initial examination of 1,877 employed middle-aged men who were free of coronary heart disease (CHD). Ten-year incidence of major CHD events (myocardial infarction and CHD death) was lowest in the first quintile of the Ho scale's distribution, highest in the middle quintile, and intermediate in the other three quintiles. The Ho scale was positively associated with crude 20-year mortality from CHD, malignant neoplasms, and causes other than cardiovascular-renal diseases and malignant neoplasms. After adjustment for risk factors, the Ho scale had a statistically significant, positive, monotonic association with 20-year risk of death from all causes combined. A difference of 23 points on the Ho scale was associated with a 42 percent increase in the risk of death. Results suggest that the Ho scale may be associated with factors having broad effects on survival. (Auth. Abs. Mod.)

84–0189. Thomas, J.; Neser, W. B.; Thomas, J.; Semenya, K.; Green, D. R. **Precursors of Hypertension: A Review.** *Journal of the National Medical Association* 75(4): 359-369, April 1983.

Data indicate that heavy smoking is one of several predisposing or enhancing factors in hypertension development. Investigators agree that smoking causes blood pressure to rise acutely in both normal and hypertensive patients and impedes therapeutic control of patients with hypertension. Both cigarette smoking and caffeine consumption cause short-lived elevation of blood pressure (2 to 120 minutes). A chain smoker could maintain elevated pressure (10 to 15 mmHg above the usual level) for several hours/day, thereby contributing to sustained hypertension. It is suggested that an increase in circulating adrenergic products following smoking may be due to norepinephrine released locally from adrenergic axon terminals within the tissues. Precursors of hypertension discussed include early elevated causal systolic blood pressure, positive family history, and obesity. Enhancing factors include high sodium ingestion and high socioecologic stress. Existence of a high-risk hypertensive personality is not well documented.

84–0190. Velican, C.; Velican, D. **Progression of Coronary Atherosclerosis From Adolescents to Mature Adults.** *Atherosclerosis* 47(2): 131-144, May 1983.

Light-microscopic examination of coronary artery samples from 222 males who had died in violent accidents (150 smokers and 72 nonsmokers, aged 16 to 45 years) revealed statistically significant ($p<0.01$) differences for atherosclerotic plaques and intimal necrotic areas, as well as incorporated microthrombi. Data were divided into six 5-year age groups: 16 to 20, 21 to 25, 26 to 30, 31 to 35, 36 to 40, and 41 to 45 years. Ten selected topographic sites of the coronary arteries were sequenced according to age, sex, branching anatomical pattern, and smoking habit, to clarify the pattern and progression of early atherosclerotic lesions from adolescence to mature adulthood. Reconstruction of thousands of static views revealed an age-related rate of progression of fibromuscular plaques, intimal necrotic areas, incorporated microthrombi, and fatty streaks. Among heavy cigarette smokers, the number of sites with incorporated microthrombi increased up to 3.6 times from one 5-year age group to the next, compared with 1.4 times in nonsmokers. Similar differences between heavy smokers and nonsmokers were detected regarding incorporation of microthrombi within fibronecrotic and fibrohyaline plaques. The fatty streak appeared as the most stable lesion related to atherogenic influences exerted by the risk factors investigated, with the exception of aging. Data, collected and analyzed for 134 females, revealed that female subjects developed the first fibromuscular plaques and intimal necrotic areas a decade later than males of similar age, and, up to age 40, showed fewer and less severe atherosclerotic lesions.

84–0191. Whitaker, N.H.G.; Spilling, R.A.E. **Evaluation of the RCGP Report on the Prevention of Arterial Disease.** *Journal of the Royal College of General Practitioners* 33(248): 135-136, 138-139, March 1983.

Control of smoking was one of four main recommendations in the Royal College of General Practitioners' 1981 report on the prevention of arterial disease. The others were (1) control of known hypertensives, (2) case finding of new hypertensives, and (3) control of known diabetics. In this pilot study of 349 patients, an average of 7 minutes/patient was spent in carrying out the recommendations. It was found that 86 were smokers, 94 ex-smokers, and 169 had never smoked. Reasons for quitting among ex-smokers included health (52 percent), expense (17 percent), both reasons (10 percent), and pregnancy (11 percent of female ex-smokers). When asked to name diseases caused by smoking, the average number mentioned correctly was 1.8 per patient (6 percent of patients did not name any). While case finding for risk factors was considered worthwhile, unless smokers can be persuaded to stop smoking, there is little point in establishing which patients are at risk. It has been shown, however, that doctors can help smokers quit. The recommendations were easily implemented and could be handled by a suitably trained nurse if necessary.

PREGNANCY AND INFANT HEALTH

84–0192. Andersen, A. N.; Lund-Andersen, C.; Henriksen, E.; Vogelius, J. **Cigaretrygnings Indflydelse pa Graviditet, Fodsel, Amnings- og Amenoreperiode. [Influence of Cigarette Smoking on the Course of Pregnancy, Delivery, Lactation and Postpartum Amenorrhoea.]** *Ugeskrift for Laeger* 144(28): 2115-2118, July 12, 1982, Danish.

Retrospective analysis of 649 women (539 smokers, or 83 percent) followed longitudinally through pregnancy and for 9 months after delivery, associated maternal smoking with significant reduction in gestational age, birth weight, birth height, and birth weight/birth height ratio; increased incidence of intrauterine growth retardation and cesarean section; and decreased incidence of preeclampsia. The birth weight/birth height ratio normalized in women who stopped smoking prior to delivery. Percentages of women who smoked before pregnancy, during pregnancy, and during lactation were 51, 43, and 41, respectively. Maternal age and early mother/infant eye contact significantly influenced duration of lactation, while an inverse relationship was noted between cigarette consumption and the duration of both lactation and postpartum amenorrhea. Cessation of smoking during lactation was not associated with significant increase in duration of breast feeding. Also discussed is the possible physiological basis (i.e., low serum prolactin) for early weaning among smokers.

84–0193. Black, P. **Smoking During Pregnancy—Are Low Tar Cigarettes Safer?** *Nursing Mirror* pp. 24-27, May 18, 1983.

Smoking low-tar cigarettes during pregnancy does not reduce fetal risk, since the main constituents affecting the fetus are nicotine and carbon monoxide. Switching to low-tar cigarettes can reduce danger of diseases caused by tar (such as carcinoma), and may benefit the nonpregnant smoker if the number of cigarettes smoked is not increased to maintain the desired nicotine level. However, smoking low-tar cigarettes during pregnancy may actually increase fetal risk, due to varying relationships between tar, nicotine, and carbon monoxide. The most common effect is low birth weight (possibly attributable to vasoconstrictive effects of nicotine or to hypoxia from carbon monoxide). The average reduction of weight (200 g) is related to the number of cigarettes smoked/day. Data for 367 mothers (143 smokers) showed highly significant differences in birth weight reduction (186 g for babies of women who smoked <15 cigarettes/day, and 225 g for babies of those smoking ≥ 15 cigarettes/day). Tabulations are given for birth weight and total tar, nicotine, and carbon monoxide consumption during pregnancy; birth weight and low-tar, low-nicotine, low-carbon monoxide cigarettes; birth weight and number of cigarettes/day; and differences between middle-tar and low-tar cigarette yields. Health education approaches for pregnant women are suggested.

84–0194. Copius Peereboom-Stegeman, J.H.J.; Van der Velde, W. J.; Dessing, J.W.M. **Influence of Cadmium on Placental Structure.** *Ecotoxicology and Environmental Safety* 7(1): 79-86, February 1983.

Preliminary results of exposing gravid rats to 0.2 mg $CdCl_2$/kg body weight/day (by subcutaneous injection) indicate that the volume density of the blood vessels in the placental stroma is reduced. Placentas of (1) 10 rats smoking 15 to 25 cigarettes/day, and 20 consuming 20 to 60 cigarettes/day, and (2) 30 nonsmoking rats were then investigated by light microscopy, and cadmium content determined by atomic absorption spectrophotometry. The cadmium content of placentas from smokers was found to be elevated compared with nonsmokers. Also, the volume density of the vascular bed in the terminal villi of the placentas of smokers was found to be decreased. It is suggested that cadmium intake via cigarette smoke provokes morphological alterations in the vascular system of placental villi in smokers. (Auth. Abs. Mod.)

84–0195. Forss, M.; Lehtovirta, P.; Rauramo, I.; Kariniemi, V. **Midtrimester Fetal Heart Rate Variability and Maternal Hemodynamics in Association With Smoking.** *American Journal of Obstetrics and Gynecology* 146(6): 693-695, July 15, 1983.

The midtrimester fetus demonstrated the narcotic, but not the hypoxic, effect of maternal smoking observed in the third trimester fetus. Maternal heart rate (MHR), blood pressure (BP), the differential index (DI) describing the short-term component of fetal heart rate (FHR) variability, and the interval index (II) describing the long-term component were measured in eight subjects in the midtrimester before, during, and after the mothers smoked a standard cigarette. The analyses of FHR variability were performed by an "on-line" method with an abdominal fetal electrocardiogram used as a triggering signal and a sample time of 1 minute. An increase of MHR, FHR, and BP with a concomitant decrease of II was observed. Unlike findings during the third trimester, no significant change of DI was observed. Correlation analysis revealed fetomaternal hemodynamic relationships different from those in the third trimester: a negative correlation between DI and FHR ($p<0.01$) and between DI and MHR ($p<0.01$). There was a positive correlation between FHR and MHR ($p<0.001$). Furthermore, no correlation was found between DI and BP or between II and MHR. (Auth. Abs. Mod.)

84–0196. Golding, J.; Butler, N. R. **Maternal Smoking and Anencephaly.** *British Medical Journal* 287(6391): 533-534, August 20, 1983.

Incidence of anencephalic births among mothers who smoked at start of pregnancy was calculated at 2.89/1,000, compared with 2.18/1,000 births ($p<0.05$) in nonsmoking mothers. However, in this sample, incidence of anencephaly rose sharply as social class fell: 1.42 in classes I and II, 2.39 in class III, and 3.16 in social classes IV and V. The proportion of mothers who smoked followed the same trend, and standardization for the social classes resulted in no significant differences between the observed and the expected in each smoking category. Data suggest that while smoking is correlated with reduced birth weight and perinatal loss, central nervous system defects show no association.

84–0197. Gunn, T. R.; Fisher, A.; Lloyd, P.; O'Donnell, S. **Antenatal Education: Does It Improve the Quality of Labour and Delivery?** *New Zealand Medical Journal* 96(724): 51-53, January 26, 1983.

Smoking correlated with nonattendance at prenatal education classes (but also with easier second stage labor) among 196 nulliparous women who delivered over a 2-month period in West Aukland, New Zealand. The 78 percent who attended the classes (39 percent smokers) had a significantly longer second stage during labor and required more forceps assistance during delivery compared with nonattenders. Fewer of the mothers who did not attend prenatal classes were married and were more often Maori and Pacific Island mothers, were younger, smoked more (70 percent were smokers), and had a lower socioeconomic status. There was no difference in the type of pain relief required during childbirth by the attenders or nonattenders. These results remained for the Polynesian mothers when analyzed separately, and differences in age and socioeconomic status were no longer present. It is noted that infants of the nonattenders were 176 g lighter, but the difference was not significant. (Auth. Abs. Mod.)

84–0198. Herrmann, U. **Rauchen und Schwangerschaft. [Cigarette Smoking During Pregnancy—A Review.]** *Therapeutische Umschau/Revue Therapeutique* 40(2): 108-116, February 1983, German.

There is sufficient evidence to show that smoking leads to increased perinatal mortality, and to conclude that physicians must inform pregnant women about the dangers of smoking and encourage cessation or a reduction in tobacco consumption. The hazards of maternal smoking are reviewed specifically with respect to nicotine, cyanide, cadmium, spontaneous abortion, deformity, problems of delivery, fetal adaptation to the extrauterine environment, perinatal mortality, and the cardiovascular system of the infant. The smoking habits of fathers are also discussed. (Auth. Abs. Mod.)

84–0199. Langford, E. R.; Thompson, E. G.; Tripp, S. C. **Smoking and Health Education During Pregnancy: Evaluation of a Program for Women in Prenatal Classes.** *Canadian Journal of Public Health* 74(4): 285-289, July-August, 1983.

Pregnant women smokers who had been given educational materials about smoking and pregnancy were significantly more likely to be nonsmokers 1 year after delivery than controls. Questionnaires on smoking beliefs and behavior were given to women in the 7th month of pregnancy attending prenatal classes. Subjects were divided into (1) experimental group I, receiving antismoking information and a pamplet ($n=40$); (2) experimental group II, receiving the same materials and a followup home visit ($n=37$); and (3) controls who received regular prenatal class information. All women completed questionnaires before the educational intervention, 4 months after delivery, and 1 year after delivery. Three-quarters of the women reported some smoking reduction or cessation during pregnancy, but no significant differences were found between the experimental and control groups. One year after delivery, however, the percentage of nonsmokers was significantly higher among the women who had received the educational program than among the control group (23.4 percent nonsmokers vs. 5.1 percent nonsmokers). The impact of the program was greatest among women whose partners were in higher occupational levels and among women who personally knew someone whose health had been adversely affected by cigarette smoking. The absence of a significant difference in smoking between the two groups during pregnancy suggests that educational programs should be carried out much earlier than the seventh month of pregnancy. The presence of a significant difference in smoking between the two groups 1 year after delivery suggests the long-term value of a prenatal program. (Auth. Abs. Mod.)

84–0200. Lumley, J.; Astbury, J. **Advice in Pregnancy: Perfect Remedies, Imperfect Science.** In: Enkin, M.; Chalmers, I. (Editors). *Effectiveness and Satisfaction in Antenatal Care.* Philadelphia, J. B. Lippincott Co. 1982, pp. 132-150.

Between 1957 and 1979, data for more than one-half million births (reported in almost 50 publications) consistently indicated that infants of women who smoked during pregnancy weighed from 175 to 200 g less than infants of nonsmokers. Some studies were large enough to control for additional factors associated with birth weight (i.e., social class, parity, maternal height and weight, maternal age, educational level, and previous low birth weight infants). Adverse fetal effects of nicotine and carbon monoxide have been documented, and smokers' infants are lighter at all gestations. Results of the British Perinatal Mortality Survey suggest that the maternal smoking relationship with fetal growth may not be a later pregnancy effect. While antismoking advice reduced maternal smoking 82 percent in the second trimester and 56 percent in the last, birth weights did not improve. However, the unreliability of smoking histories collected retrospectively is emphasized. Also discussed are associations between maternal smoking and infant mortality/spontaneous abortions (inconsistent); pregnant women's smoking-related attitudes, feelings, and beliefs; and sexual activity, alcohol consumption, maternal anxiety, diet, and exercise during pregnancy.

84–0201. Mahan, C. S. **New Strategies for Preventing an Old Problem: Low Birthweight.** *Journal of the Florida Medical Association* 70(9): 722-727, September 1983.

Smoking, low socioeconomic status, repetitive prematurity, and an unwanted pregnancy are the most frequently seen risks for preterm delivery. Smoking accounts for 4 to 14 percent of the attributable risk. The low weight of more than 60 percent of the low birth weight infants (under 2,500 g) born in Florida could be prevented with early and adequate prenatal care, excellent nutrition management and food supplementation, attention to stress management, and teaching the patient the signs of premature labor. It is noted that a frank discussion of smoking, using terms such as cyanide, nicotine, and carbon monoxide in connection with fetal growth retardation, mental retardation, stillbirth, and other outcomes, results in a high reduction or cessation rate at least during the pregnancy. Also, more attention needs to be given to family planning, including sterilization for those women at risk for giving birth to low birth weight babies. (Auth. Abs. Mod.)

84–0202. Peter, T.; Domokos, B.; Laszlo, K.; Erna, E.; Emoke, E. **Az Anya Terhesseg Alatti Dohanyzasanak Karos Hatasai: Ver Oxigen Affinitasi Vizsgalatok. [Deleterious Effects of Smoking During Pregnancy: Studies on Blood Oxygen Affinity.]** *Orvosi Hetilap* 124(33): 1979-1982, August 14, 1983, Hungarian.

The infants of 16 mothers who smoked from 1 to 40 cigarettes/day had significantly decreased birth weight and length compared with infants of 13 nonsmoking controls. Among the smokers, thiocyanate levels of maternal venous and newborns' cord blood sera were significantly higher and positively correlated. The standard blood oxygen affinity of newborns' cord blood was significantly increased in the smokers group and was positively correlated to the thiocyanate level of umbilical cord blood sera. Differences in the newborns' capillary blood standard oxygen affinity between the two groups at the ages of 3 and 5 days are given. The deleterious effects of maternal smoking on the fetus and the newborn are discussed. (Auth. Abs. Mod.)

84–0203. Rauramo, I.; Forss, M.; Kariniemi, V.; Lehtovirta, P. **Antepartum Fetal Heart Rate Variability and Intervillous Placental Blood Flow in Association With Smoking.** *American Journal of Obstetrics and Gynecology* 146(8): 967-969, August 15, 1983.

Results tend to support the hypothesis that decrease of differential index is due to fetal hypoxia (possibly mediated by intervillous placental blood flow decrease), and that decrease of interval index is a direct narcotic effect due to smoking. Intervillous placental blood flow and indices of fetal heart rate variability were measured from seven healthy pregnant women in the last trimester of pregnancy, once before and twice after smoking one cigarette. A blood flow reduction was observed in seven, a rise in five, and no change in two measurements. When intervillous placental blood flow decreased, both indices of variability decreased ($p < 0.001$); when it increased, the short-term component of fetal heart rate variability increased more significantly ($p < 0.001$) than did the long-term component ($p < 0.01$). (Auth. Abs. Mod.)

84–0204. Shuper, A.; Mimouni, F.; Merlob, P.; Zaizov, R.; Reisner, S. H. **Thrombocytopenia in Small-for-Gestational-Age**

Infants. *Acta Paediatrica Scandinavica* 72(1): 139-140, January 1983.

While smoking was not found to be more common in mothers of small-for-gestational-age (SGA) infants with neonatal thrombocytopenia (NT), the high frequency of normoblastosis and leukopenia in SGA infants supports the hypothesis of a shift of the pluripotent stem cell secondary to maternal smoking. It has previously been suggested that the chronic hypoxia associated with maternal smoking causes a shift of the stem cell in bone marrow toward an increased erythropoiesis and myelopoiesis. Maternal smoking did not differ statistically in mothers of 32 infants with NT compared with a control group without NT among 338 SGA infants examined. Toxemia was diagnosed in 35 mothers, and 27.7 percent of their infants had NT compared with only 4.6 percent among nontoxemic mothers. Other possible etiological factors were infections, polycythemia, trisomy 13, and drugs. The mechanism of NT in the SGA infant, however, is still not clear.

84–0205. Thomsen, S. G.; Isager-Sally, L.; Lange, A. P.; Saurbrey, N.; Schiolier, V. **Smoking Habits and Maternal Serum α-Fetoprotein Levels During the Second Trimester of Pregnancy.** *British Journal of Obstetrics and Gynaecology* 90(8): 716-717, August 1983.

Data indicate that maternal smoking is associated with a level of maternal serum α-fetoprotein (AFP) 20 percent higher than levels found among nonsmoking women. The influence of smoking on AFP levels at 16 weeks' gestation was examined in 120 smokers (2 to 30 cigarettes/day) and 138 nonsmokers. Median levels for smokers were 54.0 μg/l and for nonsmokers 44.3 μg/l ($p<0.001$). No difference in average body weight between the two groups was found that could account for the results. The possibility of smoking-induced increased permeability of the placental barrier is discussed briefly. (Auth. Abs. Mod.)

84–0206. Van der Velde, W. J.; Copius Peereboom-Stegeman, J.H.J.; Treffers, P. E.; James, J. **Structural Changes in the Placenta of Smoking Mothers: A Quantitative Study.** *Placenta* 4(3): 231-240, July-September 1983.

Placentas of matched pairs of smoking and nonsmoking mothers were investigated by quantitative light microscopic techniques. A smaller diameter of the villous capillaries, a decrease in vasculosyncytial membranes, and basement membrane thickening were observed in the placentas of smoking mothers. Quantitative morphometric methods showed a significant decrease in volume density of the fetal vessels in the terminal villi of the smoker's placenta. The exchange area of the smoker's placenta was shown to be decreased. The pathophysiological mechanisms leading to these changes and the possible role of toxic agents (i.e., cadmium) are discussed. (Auth. Abs.)

84–0207. Wittenberg, C. K. **Summary of Market Research for "Healthy Mothers, Healthy Babies" Campaign.** *Public Health Reports* 98(4): 356-359, July-August 1983.

A survey to determine effective health education techniques for high-risk black and Mexican American women ($n=130$) revealed that most ignored advice to quit smoking, limit alcohol consumption, exercise, vary food intake, and schedule regular prenatal checkups, believing that failure to do so would harm the mother more than the infant. Some doubted the need to avoid alcohol and tobacco due to previous uneventful pregnancies or knowledge of women who had experienced neither prenatal care nor related ill effects. Discussion sessions (totaling 200 hours) were simultaneously conducted with eight groups of black women and seven groups of Mexican American women (aged 15 to 22, or 23 to 34 years), who were currently pregnant or planning pregnancy within 2 years. All participants were of low socioeconomic status, had never worked in a health-related field, and, if Mexican American, spoke only Spanish or Spanish in preference to English. Responses from 163 health care providers, of whom 90 percent worked in public health settings, attributed failure to adopt new health patterns to skepticism, finances, traditional diets, and family patterns. Black women cited the strain of pregnancy. Although infant mortality rates (11.3 deaths/1,000 births in 1982) in the United States have been declining steadily during the last decade, the infant survival rate is lower than in 13 other countries and the mortality rate for black infants remains twice that of whites. The problems involved in reaching high-risk women are described, and methods of conveying needed health information are discussed.

See also, **84–0148**

OTHER DISEASES AND CONDITIONS

84–0208. Andriulli, A.; Masoero, G.; Amato, A.; Felder, M.; Benitti, V.; Dobrilla, G.; De La Pierre, M.; Verme, G. **Serum Immunoreactive Cationic Trypsinogen Response to Secretin in Normal Subjects.** *American Journal of Gastroenterology* 78(9): 579-583, September 1983.

Results indicate that cigarette smoking and alcohol consumption may produce biochemical abnormalities in pancreatic function before clinical evidence of pancreatic disease appears. Serum immunoreactive cationic trypsinogen (ICT) response to bolus injection of secretin (1 clinical unit/kg of body weight) was evaluated in 123 normal controls (56 nonsmokers, 67 smokers) and related to sex, age, smoking habit, and alcohol and coffee consumption. A 100 percent increase of serum immunoreactive cationic trypsinogen levels after secretin was considered a positive response (R+). When the population was considered as a whole, 55 percent of subjects proved to be R+, and the remaining were R-. In the R+ group, the serum immunoreactive cationic trypsinogen response was evident as early as 5 minutes after secretin, and after 2 minutes in the few cases in which it was evaluated. It is suggested that this direct leakage of the proenzyme from the pancreas may be due to an increased sensitivity of the pancreas to secretin. Coffee consumption and age of subjects did not affect the test. Male sex, smoking habit, and alcohol intake were associated with a significantly higher percentage of R+. (Auth. Abs. Mod.)

84–0209. Baker, K. A.; Didcock, E. A.; Kemm, J. R.; Patrick, J. M. **Effect of Age, Sex and Illness on Salt Taste Detection Thresholds.** *Age and Ageing* 12(2): 159-165, May 1983.

Smokers had higher salt detection thresholds than nonsmokers among 146 healthy subjects aged 10 to 95 years, and 43 ill elderly patients. Salt taste thresholds were measured with a forced-choice, up—down sip method. Thresholds are shown to increase log linearly with age after the age of 20. The lower thresholds of women reflect the lower prevalence of smoking. Ill patients had higher thresholds than healthy subjects of the same age that were not specifically associated with any diagnosis. Additional taste threshold measurement methods and earlier studies of taste thresholds are reviewed. (Auth. Abs. Mod.)

84–0210. Barnett, A. H.; Britton, J. R.; Leatherdale, B. A. **Study of Possible Risk Factors for Severe Retinopathy in Non-Insulin Dependent Diabetes.** *British Medical Journal* 287(6391): 529, August 20, 1983.

Hypertension was strongly associated with severe retinopathy in diabetics not dependent on insulin, but no association was found with smoking. Of the 100 subjects, 50 had developed severe proliferative retinopathy or maculopathy within 10 years of diagnosis of diabetes. The remaining 50 had no evidence of microvascular complications after more than 14 years of diabetes. Besides hypertension, female sex was also associated with retinopathy. However, no significant correlations were found with cigarette smoking, family history, body weight, or diabetic control.

84–0211. Bennett, J. R.; Buckton, G.; Martin, H. D. **Cimetidine in Gastro-Oesophageal Reflux.** *Digestion* 26(3): 166-172, March 1983.

Smokers showed greater improvement in gastroesophageal reflux than nonsmokers when treated with cimetidine. Intraesophageal pH monitoring and symptom scores were used to compare the effect of cimetidine (1 or 2 g/day) with placebo among 84 patients (26 smokers, 52 nonsmokers, and 6 with unknown smoking status). Night pain and antacid consumption were reduced by cimetidine, but other symptoms did not show significant differences. Symptomatic improvement appeared to be greater in smokers than nonsmokers. Intraesophageal pH measurements (15-hour) showed that reflux episodes were reduced in the cimetidine-treated groups. (Auth. Abs. Mod.)

84–0212. Churg, A. **Current Issues in the Pathologic and Mineralogic Diagnosis of Asbestos-Induced Disease.** *Chest* 84(3): 275-280, September 1983.

The major issue confronting clinicians and pathologists is which lung cancers can be reasonably ascribed to asbestos exposure, since many asbestos workers smoke, and there is no pathologic method of ascribing part or all of the injury to asbestos. Malignant mesothelioma is strongly associated with asbestos exposure, but not with cigarette smoke or other etiologic agents. While asbestos is associated with other cancers, the relationships are not strong when compared with those associated with smoking. A number of other issues in asbestos-related disease are reviewed, including asbestosis and benign pleural disease. In discussing small airways disease, it is noted that disease caused by asbestos, although probably detectable over the background of airways disease caused by smoking, is of questionable functional significance and should not be diagnosed as asbestosis. It is concluded that there is presently no easy scientific solution for attributing etiology of asbestos-induced cancers.

84–0213. Cummings, S. **Stress, Cigarettes, and Ulcers. (Letter).** *Gastroenterology* 85(5): 1232, November 1983.

Previous case reports that suggested a causal link between stress and peptic ulcers are criticized for not mentioning the smoking habits of patients. Smoking is a powerful risk factor for both the occurrence and recurrence of peptic ulcer, and smokers increase consumption in stressful situations. The effects of smoking on gastric acid secretion are controversial. However, the role of acid hypersecretion in causing ulcers is uncertain and thus other potential contributing factors, such as smoking, should be included in future research.

84–0214. Cunningham, D. R.; Vise, L. K.; Jones, L. A. **Influence of Cigarette Smoking on Extra-High-Frequency Auditory Thresholds.** *Ear and Hearing* 4(3): 162-165, May-June 1983.

While no statistically significant differences were found between the extra-high-frequency (EHF) thresholds of 18 smokers (mean age 28.9 years, mean consumption 1.5 packs/day for 11.1 years) compared with 25 nonsmokers (mean age 25.7 years), data indicate that smokers have less acute EHF auditory sensitivity. Differences in mean EHF thresholds for smokers vs. nonsmokers were demonstrated for only one test frequency, 9 kilohertz (kHz). However, 22 percent more smokers than nonsmokers were unable to hear the EHF stimuli at 18 kHz, and smokers' mean hearing thresholds were consistently (though not significantly) less acute. It is suggested that a similar study of older subjects might show greater EHF threshold differences, and that differences favoring nonsmokers might increase in direct proportion to subjects' age and cigarette consumption levels. Furthermore, carboxyhemoglobin should be determined for each subject and correlated with EHF thresholds. The relationship between smoking and atherosclerotic vascular disease is also discussed.

84–0215. Daniell, H. W. **Postmenopausal Tooth Loss. Contributions to Edentulism by Osteoporosis and Cigarette Smoking.** *Archives of Internal Medicine* 143(9): 1678-1682, September 1983.

Observations strongly suggest that middle-aged women may be more likely to retain their teeth if they avoid smoking and undertake a program to prevent progression of osteoporosis. Smoking habits and current osteoporosis severity (percent cortical area (PCA) at metacarpal midshaft) were compared with the age at which 208 white women (77 smokers, 131 nonsmokers, aged 60 to 69 years) had acquired 218 upper or lower full dentures. Among osteoporotic women (PCA <70 percent) who still had their natural teeth at age 50 years, 44 percent had required a new full denture before age 60 compared with 15 percent of nonosteoporotic women (PCA >80). Different denture requirements between these groups had not existed before age 50, but continued after age 60. Fifty-two percent of smokers, 26 percent of nonsmokers, and only 8 percent of nonosteoporotic nonsmokers had required dentures after age 50. (Auth. Abs. Mod.)

84–0216. Ibrahim, A. S.; Fatt-Hi, A. S. **Cigarette Smoking and Hearing Loss.** *International Journal of Health Education* 2(2): 31-33, July 1983.

Comparison between 150 smokers (average age 42.9 years) and 150 nonsmokers (average age 43.4 years) associated smokers with significantly (1) lower incidence of mild grade hearing loss; (2) higher incidence of both conductive and perceptive deafness; and (3) higher average hearing loss. Hearing loss was significantly correlated with age ($p=0.001$) for both smokers and nonsmokers. Among smokers, hearing loss was also significantly correlated with duration of smoking ($p=0.01$). Since duration of smoking was also correlated with age ($p=0.01$), partial correlation analysis was performed. Results showed that duration of smoking had a greater (and independent) effect on hearing loss than age. Possible mechanisms by which smoking increases incidence and severity of both conductive and perceptive deafness are suggested.

84–0217. Ippoliti, A.; Elashoff, J.; Valenzuela, J.; Cano, R.; Frankl, H.; Samloff, M.; Koretz, R. **Recurrent Ulcer After Successful Treatment With Cimetidine or Antacid.** *Gastroenterology* 85(4): 875-880, October 1983.

The percentages of ulcer patients who had a recurrence at 6 months after 6 weeks of treatment were highest for smokers (≥ 0.5 pack/day), those with a disease duration of more than 4 years, and those with a family history of ulcers. Healing rates were also lower among all smokers compared with nonsmokers, emphasizing the influence of variables other than treatment. Of the 83 patients treated with Mylanta, 58 percent were smokers; for the 78 on cimetidine treatment, 60 percent were smokers. The rates of healing and recurrence after treatment with cimetidine or antacid were compared in 138 patients with endoscopically documented duodenal ulcer, in a randomized, double-blind trial. Healing rates under both treatments were almost identical, and no differences in the frequency of recurrences were found. Other variables associated with delayed healing or recurrence included sex, pain frequency, and acid secretion.

84–0218. Ismail, A. I.; Burt, B. A.; Eklund, S. A. **Epidemiologic Patterns of Smoking and Periodontal Disease in the United States.** *Journal of the American Dental Association* 106: 617-621, May 1983.

Current smokers had higher mean periodontal index (PI) scores and significantly higher scores on the debris, calculus, and oral hygiene indexes ($p<0.005$) than nonsmokers among 20,749 respondents to the National Health and Nutrition Examination Survey (NHANES). The association between poorer levels of periodontal health and smoking remained even after accounting for age, sex, race, oral hygiene, socioeconomic status, and frequency of daily toothbrushing variables. When analysis was restricted to smokers only, no clear-cut association was demonstrated between periodontal disease and the duration of smoking or the number of cigarettes smoked/day. These results suggest that dental practitioners should discourage patients from smoking for reasons of periodontal health as well as general preventive care. (Auth. Abs. Mod.)

84–0219. Journal of Traffic Medicine. **Smoking in the Car.** *Journal of Traffic Medicine* 11(2): 36, 1983.

Smoking in closed cars poses both immediate and indirect dangers. Sidestream smoke contains five times the carbon monoxide (CO), three times the tar and nicotine, four times the carcinogens, and 46 times the ammonia of the mainstream smoke. Nonsmokers can thus be exposed to a saturated atmosphere of sidestream smoke inside a closed car. Temporary poisoning by CO produces delayed reflexes, miscalculation of distance, and impairment of night vision. In addition, the manipulation of smoking materials and dropping of hot ash can impair driving performance.

84–0220. Kellow, J. E.; Barr, G. D.; Middleton, W.R.J.; Piper, D. W. **Comparison of Colloidal Bismuth Subcitrate Tablets and Liquid in Duodenal Ulcer Healing.** *Journal of Clinical Gastroenterology* 5(5): 417-420, October 1983.

Smoking showed no significant impact on ulcer healing rates in 51 outpatients (28 smokers, 23 nonsmokers). A double-blind trial, with endoscopically proven duodenal ulcer patients, compared the effectiveness of colloidal bismuth subcitrate (CBS) in tablet vs. liquid form in the healing of duodenal ulcers. Patients were given either one tablet four times/day, or 5 ml liquid four times/day for 4 weeks. Ulcer symptoms and their relief were recorded by patients, with data on cigarette, alcohol, and drug intake. Endoscopy performed after 4 weeks showed that 18 of 26 patients taking tablets (69 percent) and 19 of 25 patients taking liquid (76 percent), had healed ($p=0.82$). Symptomatic improvement was similar with both forms of CBS. Smoking and analgesic ingestion did not influence healing rate. It is concluded that CBS tablets and liquid are equally effective in healing duodenal ulcer. (Auth. Abs. Mod.)

84–0221. Keys, A. **Smoking, Lung Function, and Body Weight. (Letter).** *British Medical Journal* 286(6380): 1823-1824, June 4, 1983.

While previous writers (*British Medical Journal* 286(6362): 249-251, January 22, 1983; *British Medical Journal* 286(6373): 1280, April 16, 1983) have focused attention on lung function to explain the lower weight of smokers compared with nonsmokers, it is suggested that appetite and oral satisfaction may play a significant role. Among healthy businessmen who voluntarily stopped smoking, average weight gain after 2 years was 3.7 kg compared with 0.5 kg in a matched group who continued to smoke. While the relationship between relative weight and smoking is still unclear, smoking tends to depress the appetite and sense of taste, suggesting that the inverse relationship is closer to the gastrointestinal system than to the respiratory system. See also 83-1188, 84-0155.

84–0222. Korman, M. G.; Hansky, J.; Eaves, E. R.; Schmidt, G. T. **Influence of Cigarette Smoking on Healing and Relapse in Duodenal Ulcer Disease.** *Gastroenterology* 85(4): 871-874, October 1983.

Smoking influenced the short-term healing and relapse rate in 135 patients (71 smokers) with duodenal ulcer who were treated with cimetidine (90), ranitidine (25), and oxmetidine (20), all powerful H_2-receptor antagonists. Ulcer healing and relapse were documented endoscopically and all studies were performed in a double-blind manner. In the short term, 95 percent of nonsmokers healed compared with 63 percent of smokers ($p<0.01$), and there was a positive correlation between failure to heal and number of cigarettes smoked. During a 12-month followup examination after healing and on no treatment, 53 percent of nonsmokers and 84 percent of smokers relapsed ($p<0.01$). Results show that smoking adversely affects healing of duodenal ulcer with H_2-receptor antagonists, and that continued smoking leads to a higher relapse rate. (Auth. Abs. Mod.)

84–0223. Kowolik, M. J.; Nisbet, T. **Smoking and Acute Ulcerative Gingivitis.** *British Dental Journal* 154(8): 241-242, April 23, 1983.

Smoking, while established as a consistent contributor to acute ulcerative gingivitis (AUG), is not an overriding influence in the absence of other factors. The average time interval between smoking initiation and AUG was approximately 6 to 8 years, but the range among the 100 clinic patients (98 smokers) was wide and extremely variable. The range in patients (58 female and 42 male, average age 23.1 years), with severe disease ($n=22$) was from 1.5 to 20 years. Although only broad categories for consumption levels were used, no positive correlation was found between the quantity of tobacco consumed and disease severity. Most disease occurred during the winter months (82 percent of severe cases). The low proportion of smokers ($n=37$) in the matched group of 100 controls with chronic gingivitis, who were also attending the clinic for treatment, may have been due to a discrepancy between the groups in terms of socioeconomic status; controls attending the clinic were more motivated toward general health care. While the precise etiology of AUG remains obscure, there is undoubtedly an association with poor oral hygiene, emotional stress, crowded living conditions, and possibly debilitating illness. Any reduction in AUG may be a function of improvement in any or all of these factors, as well as a reduction in the number of smokers.

84–0224. Layde, P. M.; Peterson, H. B.; Dicker, R. C.; DeStefano, F.; Rubin, G. L.; Ory, H. W. **Risk Factors for Complications of Interval Tubal Sterilization by Laparotomy.** *Obstetrics and Gynecology* 62(2): 180-184, August 1983.

Smoking was one of four major risk factors for complications following interval tubal sterilization in 282 women undergoing laparotomy. The relative risk was highest in women with diabetes mellitus (4.1), followed by smoking (2.4), history of pelvic inflammatory disease (2.2), and previous abdominal or pelvic surgery (1.8). Using a standard definition of major complications, the overall complication rate was 5.7/100 procedures. Women experiencing complications had a significantly lengthened postoperative recovery period before the resumption of normal activities. Women with an initial abdominal incision of 7 cm or longer had three times the complication rate of women with shorter incisions. Results provide objective evidence that, for tubal sterilizations, minilaparotomy (laparotomy with a small abdominal incision) is associated with lower morbidity than conventional laparotomy. (Auth. Abs. Mod.)

84–0225. Nasrallah, S. M.; Martin, D. M. **Serum Isoamylase as a Test for Pancreatic Insufficiency.** *Gut* 24(2): 161-164, February 1983.

Data indicate that total amylase (T), pancreatic amylase (P), or the P/T ratio, are not influenced by smoking or meal stimulation. Total and pancreatic serum isoamylases were performed on 38 control subjects (21 nonsmokers, 17 smokers) and 21 consecutive patients with pancreatic insufficiency before and after meal stimulation. There were no differences in the fasting or stimulated levels of T, P, or P/T between smokers and nonsmokers. The P/T ratio was significantly reduced in patients with pancreatic insufficiency when compared with control groups ($p<0.0005$); 11 of 21 patients with pancreatic insufficiency had raised total serum amylase, but none had a rise in the pancreatic fraction or P/T ratio. (Auth. Abs. Mod.)

84–0226. Rantakallio, P.; Makinen, H. **The Effect of Maternal Smoking on the Timing of Deciduous Tooth Eruption.** *Growth* 47(2): 122-128, Summer 1983.

Tooth eruption was advanced by an average of 1 week in 1,559 children of mothers who smoked during pregnancy. Followup investigation of pregnant women since 1966 has provided data on 10,443 children. The smoking habits of 9,981 mothers were known: 8,422 were nonsmokers; 1,303 smoked < 10 cigarettes/day; and 256 smoked ≥ 10 cigarettes/day. Children of the smokers have been found to have poorer health, delayed development, and increased mortality at all ages compared with children of matched controls. It is therefore surprising that maternal smoking advances tooth eruption. Data give no indication for the positive correlation, but maternal smoking may affect development rate of deciduous teeth or their penetration through the gum, or may exercise an effect through lactation. Other possibilities are also discussed. Other significant variables were weight and height at birth and 6 months, and gestational age.

84–0227. Repace, J. L. **Risks of Passive Smoking.** Center for Philosophy and Public Policy, University of Maryland, College Park, Maryland, Working Paper RC-8, August 1983, 31 pp.

The risks to nonsmokers from breathing indoor air pollution from tobacco smoke (passive or involuntary smoking) are discussed from medical, legal, social, historical, and philosophical points of view. Problems encountered by nonsmokers who are hypersensitive to tobacco smoke are contrasted with chronic risks to normal nonsmokers. Ambient tobacco smoke is compared with hazardous outdoor air pollutants and the products of other human activities that are regulated by society. It is argued that failure to regulate indoor smoking will invite increasing confrontation and social dissension between nonsmokers, smokers, and employers, since the nonsmoker's right to clean indoor air is morally superior to the smoker's right to pollute. Legal strategies to remedy the situation are outlined. (Auth. Abs. Mod.)

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

84–0228. Ahmed, E. N.; Jafarey, N. A. **Smoking Habits Amongst Medical Students of Sind Medical College.** *Journal of the Pakistan Medical Association* 33(2): 39-44, February 1983.

Pleasurable relaxation and tension reduction were the reasons given most often for smoking by the 191 medical students (188 males, 3 females) who smoked. Results of a questionnaire survey, conducted from January to July 1981, showed that the majority of students were nonsmokers (n = 1,145, 668 males and 477 females) and a few were ex-smokers (26 males, 1 female). The average age of initiation of smoking was 17 years for smokers and 18 years for ex-smokers. The six students classified as heavy smokers (≥ 30 cigarettes/day) were in the top two (most senior) classes. Among ex-smokers, 81 percent had smoked 10 or less cigarettes/day, compared with 70 percent among smokers. Most (63 percent) had quit smoking when they realized its adverse health effects, and 52 percent also felt it was a waste of money. Of the 481 female students, 477 (99.19 percent) were nonsmokers. Brothers, but not fathers, were an important family influence in preventing or encouraging a person to smoke. It is concluded that smoking is a psychological habit with pharmacological and social satisfactions and reinforcements. For successful cessation, a person must want to give up smoking.

84–0229. Aneshensel, C. S.; Huba, G. J. **Depression, Alcohol Use, and Smoking Over One Year: A Four-Wave Longitudinal Causal Model.** *Journal of Abnormal Psychology* 92(2): 134-150, May 1983.

Over the long term (1 year), smoking level was very stable and neither influenced, nor was influenced by, depression. Longitudinal data collected at four time periods from an adult community sample (n=742) were used to study the relationships between smoking, alcohol use, and depression (measured by survey interviews) in a latent variable causal model. Current levels of depression were moderately stable over the 1-year period. In the short term, depression was related to a slight increase in alcohol use, and alcohol use was related to a slight decrease in depression, but in the long run alcohol was related to increased depression. Smoking level was very stable over both the short and long run.

84-0230. Breed, W.; De Foe, J. R. **Cigarette Smoking on Television, 1950-1982.** *New England Journal of Medicine* 309(10): 617, September 8, 1983.

Analysis of the extent to which changes in programing reflect scientific findings on smoking and health showed an approximate 10-fold drop in frequency of cigarette smoking on prime-time television. Four coders monitored reruns of situation comedies and 1-hour dramas on 24 stations in four States, obtaining a sample of 280.5 hours representative of a 32-year period: before and after the Surgeon General's 1964 report on smoking and health; before and after the 1971 ban on televised cigarette commercials; and the 1981-1982 television season. Effects of the Surgeon General's report appeared to be more related to situation comedies, while the advertising ban effects seemed more related to dramas. While characters from all occupational strata smoked, changes in the medical profession were particularly noteworthy. In a 1961 episode of *Dr. Kildare*, both Dr. Kildare and his mentor, Dr. Gillespie, smoked; however, no cigarettes were seen in 10 hours of two 1971-1977 hospital dramas (*Medical Center* and *Marcus Welby, M.D.*). Tabulated data for average smoking frequency/hour by program type are provided. Parallel data on alcohol consumption showed increases over the four periods.

84-0231. Canadian Family Physician. **18-19-Year-Old Women Are Smoking Up a Storm.** *Canadian Family Physician* 29: 1498, August 1983.

Results of the 1981 Canada Fitness Survey show that smoking is more prevalent among females than males in every age group. Of 14- to 15-year-olds, 11 percent of males and 17 percent of females smoke, while rates for 16- to 17-year-olds increase to 29 percent of males and 34 percent of females. Of 18- to 19-year-olds, 37 percent of males smoke, compared with 46 percent of females (exceeding the 40 percent national average). More than 22,000 Canadians were interviewed and about 16,000 took fitness tests. Of children and youth, 71 percent are physically active, compared with 56 percent of the entire Canadian population. Also provided are alcohol consumption statistics by age.

84-0232. Duryea, P. E.; Kreuter, M. W.; Braza, G. F. **Cognitive Perceptions of Importance in Students' Decisions About Smoking.** *Health Education* pp. 4-8, September-October 1981.

As grade level increases, health concerns decrease while the importance of social factors becomes magnified in a student's decision to smoke. The open-ended questionnaire, administered to 726 students in the 5th, 7th, 9th, and 11th grades, was designed to elicit the key factors a student considers in making a smoking decision. The questions were subdivided into two categories designated "health concerns" and "social concerns." In both the "yes" and "no" intent-to-smoke groups, social concerns increased with grade level, while health concerns declined. However, in the "yes" group, social concerns were initially higher, and remained higher, than health considerations. Among students who were not sure of their intentions, no definitive pattern was observed. The mean number of cognitive categories considered in the decision was not affected by grade level. Data suggest that the subjects who chose not to smoke or who were undecided considered a wider range of value categories in their decision than the "yes" individuals.

84-0233. Eastwood, M. R. **Smoking Profiles of Patients Admitted for Neurosis. (Letter).** *British Journal of Psychiatry* 142: 206, February 1983.

A response prompted by the authors' reply to criticism of methods and conclusions reported in a previous article (*British Journal of Psychiatry* 139: 43-46, July 1981) suggests that absence of a significant association between smoking and neuroticism previously found in larger community samples should be regarded more seriously than generalizations derived from data from a small and select inpatient sample. The absence of a significant difference between groups in the younger age category appears to weaken the argument that neurotics begin smoking at an earlier age. Furthermore, of 17 statistical comparisons reported, only 4 resulted in values generally accepted as reasons to reject the null hypothesis. It is stated that smoking is too important a public health problem for associative or etiological factors to be imputed on scant evidence. See also 81-1907, 82-1099, 84-0258.

84-0234. El Maksoud, E. A.; Cappiello, L. A. **A Profile Study of High School Boys Who Smoke Cigarettes in Alexandria, Egypt and Buffalo, New York.** *International Journal of Health Education* 2(1): 38-40, March 1983.

Smoking profiles of high school boys in Alexandria, Egypt, and Buffalo, New York, were compared. In Alexandria, 192 of 573 boys smoked (47.4 percent less than five cigarettes/day and 5.2 percent more than a pack/day). In Buffalo, 113 of 424 boys smoked (32.9 percent less than five cigarettes/day and 24.8 percent over a pack/day). Although the Buffalo group had more smokers and more heavy smokers, the smokers were more apt to think that they could stop smoking permanently (81.4 percent compared with 50.5 percent) and more of them had tried to quit (66.4 percent compared with 44.8 percent). In Buffalo, more parents and teachers knew about the boys' smoking. Smoking by fathers was about the same, but the Buffalo group had a much higher percentage of mothers who smoked (61.1 percent) than the Alexandria group (16.6 percent). Mothers in Buffalo were also more likely to punish their sons for smoking. While the percentage of Buffalo students who reported feeling the need to smoke was more than twice that of Alexandria students, 83 percent of Buffalo boys and 38 percent of Alexandria boys reported that they did not know why they smoked.

84–0235. Fox, B. **Will Snuff Sniffing Be the Next Youth Craze?** *New Scientist* 99(1369): 330, August 4, 1983.

While 10 million adults have quit smoking over the past 10 years, the tobacco companies are reintroducing snuff in campaigns directed primarily toward youngsters. Both the Health Education Council and Action on Smoking and Health have warned that snuff may be a risk factor for nose cancer. However, a recent free offer from a subsidiary of the Imperial Tobacco group attempted to promote snuff use among the predominantly youthful readers of a pop music publication. The offer, which included a free box of snuff, instruction booklet for beginners, and history of snuff sniffing, was supposedly limited to those over age 18; however, there was no way to verify the age of respondents. It is suggested that, like cigarette smokers, snuff sniffers may become statistics.

84–0236. Genedani, S.; Bernardi, M.; Bertolini, A. **Sex-Linked Differences in Avoidance Learning in the Offspring of Rats Treated With Nicotine During Pregnancy.** *Psychopharmacology* 80(1): 93-95, 1983.

Although avoidance learning was similar in both male and female control groups, nicotine exposure during fetal life improved learning in females but reduced it in males, showing a statistically significant difference from day 17 until day 25 (end of training period). The offspring of rats treated subcutaneously with nicotine, 0.5mg/kg/day on days 1 to 20 of gestation, were trained for active avoidance conditioning when 60 days old. Nicotine had no significant effects on litter size, weight of viable fetuses, total number of dead fetuses, or weight gain of mothers during pregnancy. No gross malformations were observed. (Auth. Abs. Mod.)

84–0237. Grout, P.; Cliff, K. S.; Harman, M. L.; Machin, D. **Cigarette Smoking, Road Traffic Accidents and Seat Belt Usage.** *Public Health* 97(2): 95-101, March 1983.

Case—control data (114 cases, 310 controls) show that smokers are more likely to experience injury-producing traffic accidents at night, and are significantly less likely to wear seat belts, compared with nonsmokers. While the overall percentage of nighttime accidents (30 percent) differed little between cases and controls, data for smoking and alcohol consumption indicated that 20 percent of nonsmokers and 45 percent of smokers were involved in accidents at night. Corresponding figures for occasional and regular drinkers were 23 and 38 percent, respectively. Among cases and controls, the number of subjects who always wore seat belts was similar (31 percent); however, only 18 percent of smokers always wore seat belts, compared with 41 percent of nonsmokers (significant). Of smokers, 28 percent approved of seat belt legislation, compared with 40 percent of nonsmokers (significant). The relationship between smoking and accident involvement is discussed with regard to (1) physical factors that require the driver to concentrate on a task other than driving (i.e., lighting, holding, and inhaling the cigarette), or that impair vision (i.e., coughing and watery eyes, or a smoky atmosphere and deposits on windows); (2) physiological factors that may reduce night vision, or decrease visual perception as smokers' carboxyhemoglobin levels increase; and (3) psychological factors, such as personality differences and reduced probability of seat belt use.

84–0238. Hamermesh, D. S.; Hamermesh, F. W. **Does Perception of Life Expectancy Reflect Health Knowledge?** *American Journal of Public Health* 73(8): 911-914, August 1983.

White male smokers in two surveys estimated their longevity as 4 years less than that of nonsmokers, roughly the actuarial difference. The first survey was done on 822 economists aged 26 to 65; the second on 726 men living in the Midwest, aged 20 to 70. Those reporting that they did not exercise expected to live as long as those who did exercise, somewhat inconsistent with available information. Men with long-lived parents overestimated their longevity, and those more than 15 percent overweight underestimated their life expectancy, in terms of actuarial figures. Results suggest that antismoking campaigns have made smokers aware of the detrimental effects of smoking on longevity.

84–0239. Harris, J. E. **Cigarette Smoking Among Successive Birth Cohorts of Men and Women in the United States During 1900—80.** *Journal of the National Cancer Institute* 71(3): 473-479, September 1983.

For each birth cohort, recent smoking cessation rates for men exceeded those for women. Maximum exposure to cigarette smoking probably occurred among men who are now in their seventh and eighth decades. By contrast, peak exposure to smoking probably occurred among women who are now only in their fifth and sixth decades. Trends in cigarette smoking among successive cohorts of men and women were reconstructed from smoking histories of respondents to the 1978 to 1980 Health Interview Surveys (20,440 males, 24,955 females). Estimated smoking rates among the older cohorts were then adjusted for the differential mortality of cigarette smokers and nonsmokers. While over half the young male population were smoking cigarettes by 1920, the proportion of young female smokers did not exceed one-third until 1950. The proportion of men who ever smoked cigarettes progressively declined with each successive cohort born after 1920. By contrast, the proportion of women who ever smoked declined only among cohorts born after 1940. Cessation of cigarette smoking after age 30 was observed for all cohorts of men, and rates accelerated for men who reached age 30 after 1960. By contrast, smoking cessation from age 30 was observed only for women who reached age 30 after 1950. Cessation rates accelerated only for women who reached age 30 after 1970. (Auth. Abs. Mod.)

84–0240. Harris, M. **How Young People See Things.** *New Society* 65(1078): 54, July 14, 1983.

An independent survey by the Department of Education and Science revealed that, although two-thirds of British 14- to 19-year-olds completely reject all drugs, alcohol and tobacco (associated with adult status) are still eagerly consumed. Expenditures for cigarettes rivaled those for alcohol, but outranked books, concerts, football matches, and sports equipment. A third of the males over 17 years of age drank alcohol regularly, and over half occasionally. Overall, the data reflect a faith in the status quo, echoing attitudes in the adult population and existing legislation. Other attitudes reported include the perceived causes of urban rioting, relationships with the opposite sex, family life, and political issues.

84–0241. Jobe, J. B.; Holgate, S. H.; Scrapansky, T. A. **Risk Taking as Motivation for Volunteering for a Hazardous Experiment.** *Journal of Personality* 51(1): 95-107, March 1983.

Noncommissioned officers, smokers, later-born children, and children of lower socioeconomic class parents were significantly overrepresented among Army male personnel who volunteered for a hazardous combat simulation experiment. Comparisons with nonvolunteers showed 70 percent of volunteers were smokers (42 percent of nonvolunteers), 81 percent had a lower social class father (50 percent), 79 percent were later born (50 percent), and 63 percent were noncommissioned officers (33 percent). Results support the hypothesis that smoking is a measure of arousal-seeking behavior due to its nature as a central nervous system stimulant. Subjects were given a biographical and personal habit questionnaire, the IPAT Anxiety Scale, Rotter's Locus of Control Scale, and Torrance and Ziller's life experience inventory. Results indicated that volunteers were significantly less anxious and more willing to take risks than were nonvolunteers. For a routine, nonhazardous experiment, the only variable that discriminated between volunteers and nonvolunteers was mother's educational level. Results are in agreement with findings, using college students, that volunteer samples differ significantly from nonvolunteer samples, and that the characteristics that discriminate between these two groups vary as a function of situational factors. (Auth. Abs. Mod.)

84–0242. Joly, D. J.; Sarmientos Acosta, M. R. **The Cigarette-Smoking Habit Among Preuniversity Students in Havana, Cuba, in 1980.** *Bulletin of the Pan American Health Organization* 17(2): 158-163, 1983.

In a survey of 950 Cuban students in grades 10 to 12, between 40 and 50 percent of those aged 18 or over smoked at least one cigarette/day. Most of the smokers consumed four or more cigarettes/day, about 20 percent smoked 10 or more cigarettes/day, and 38 percent reported inhaling. Smoking prevalence increased rapidly between the ages of 15 and 18; 16.2 percent of those aged 15 were smokers compared with 35.8 percent of those aged 17. Overall, about one-third of the boys and one-fifth of the girls 15 to 17 years old were smokers. Smoking was found to be associated with parental smoking patterns and strongly associated with sibling smoking patterns. Data also indicated an association between cigarette smoking and beer drinking, but showed comparatively little association between student smoking and parental education level. Smokers had somewhat poorer grades, did less well in sports, and experienced a higher incidence of respiratory problems than their nonsmoking counterparts. Absenteeism was also higher among smokers, and it is suggested that these variables may be related to one or more factors such as family environment, personality, and peer group behavior. The ultimate prevalence of smoking among Cuban adults is likely to be considerably higher than that found among even the oldest students interviewed, since it has been reported previously that a large proportion of Latin American smokers do not begin smoking before the age of 20.

84–0243. Kellam, S. G.; Stevenson, D. L.; Rubin, B. R. **How Specific Are the Early Predictors of Teenage Drug Use?** In: Harris, L. S. (Editor). *Problems of Drug Dependence 1982.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, National Institute on Drug Abuse Research Monograph Series No. 43, April 1983, pp. 329-334.

Aggressiveness in first grade predicts future heavy cigarette, drug, and alcohol use as well as delinquency in males but not in females. Students from elementary schools in a black, poor, urban community, first rated in 1966-1967 (n = 1,242), were interviewed 10 years later (n = 705) to determine the social adaptation characteristics predictive of future behaviors. All predictors are behavioral responses rated by teachers or scored on tests of classroom task performance. Shyness predicts an inhibition of both substance use and delinquency, but also predicts higher anxiety levels in adolescence. First grade tests of readiness and IQ are clearly related to drug and alcohol use, but not smoking, in both males and females. The higher the child's score, the higher the probability of future use. Results indicate that (1) prevention efforts that might reduce the risk for aggressive first graders could be counterproductive for brighter children; (2) sex differences in developmental behavior patterns are important; and (3) studying one outcome in isolation from others loses much information about the meaning and function of the predictors.

84–0244. Kleinke, C. L.; Staneski, R. A.; Meeker, F. B. **Attributions for Smoking Behavior: Comparing Smokers With Nonsmokers and Predicting Smokers' Cigarette Consumption.** *Journal of Research in Personality* 17(2): 242-255, June 1983.

A 6-hour battery of personality tests (a revised version of Coan's Smoking Survey) comparing smokers (n = 214 for two studies) with nonsmokers (n = 220 for two studies) showed that smokers gave significantly more agreement than nonsmokers to only one smoking motive: relaxation. The most important motives predicting cigarette consumption were addiction and affective smoking. Nonsmokers were significantly more likely than smokers to attribute smokers' motives to sophistication, addiction, stimulation, and sensorimotor pleasure, and were more likely to perceive smoking as unhealthy and a public nuisance. Multiple correlations between smoking motives and self-reported cigarette consumption were .754 (study 1) and .670 (study 2). Results were related to attributional theories of arousal and to research measuring human (in)ability to accurately explain behavior. It was recommended that smoking cessation programs provide smokers with skills needed for relaxation and coping with withdrawal. (Auth. Abs. Mod.)

84–0245. Lamarsaude, B. **Reflexion Pour Approcher la Complexite des Mecanismes Psychologiques Sous Facents a la Dependance Tabagique. [Reflection on Approach to the Complex Psychologic Mechanisms Underlying Smoking Dependence.]** In: Molimard, R. (Editor). *1ere Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 85-90, French.

The motto "smoking is unreasonable but the problem of smoking cessation is not solved by reasoning" is the basis of a psychoanalytic approach attempting to show both the complexity of the pathologic smoker phenomenon and the difficulty of smoking withdrawal. Orality is one of many psychological aspects of individual smoking dependence admitted during psychoanalytic treatment. Gratification of smoking, compared with the oral gratification a newborn derives from breast feeding, develops into an autoerotic system that represses anxiety associated with the absence of breast feeding or its substitute (wherein a cigarette fills the same stress-relieving role as a maternal breast). This is confirmed by the depression often exhibited by a smoker in the process of withdrawal. Also, the mental state of a smoker is comparable to that of an alcoholic. Hence, the same difficulties are expected in detoxification of smokers as those associated with treatment of alcoholics, particularly the danger of relapse. It is concluded that smoking is a substitute for alcohol addiction and that it is practiced by individuals who lack the necessary boldness to gratify their needs by drinking. This observation sheds new light on the difficulties associated with overcoming tobacco dependence.

84–0246. Le Magnen, J. **Denominateurs Communs de la Dependance. Les Dependances Croisees: Tabac, Alcool, Opiaces. [Common Denominators of Dependency. Cross-dependence: Tobacco, Alcohol, Opiates.]** In: Molimard, R. (Editor). *1ere Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 61-76, French.

Current reports on analogy and differences between tobacco (nicotine), alcohol, and narcotics dependencies or habits and the possible existence of a common neuropharmacological mechanism of dependence induction are reviewed. Chronic toxicity (dependence) is considered the most important common characteristic of all studied agents called "toxicophilogenic." Empirical observations suggest that, in contrast with alcohol and narcotics, (1) smoking dependence develops very easily and rapidly, even in moderate smokers, by absorption of a low nicotine dose; (2) tobacco is as inoffensive a drug as coffee; and (3) first symptoms of smoking withdrawal appear more frequently. Toxicophobic and toxicophylic mechanisms of animal and human behavior upon ingestion of the studied toxic agents are reviewed. Acute interaction is revealed between neuropharmacological effects of (1) alcohol intake simultaneously with smoking in humans, and (2) a narcotic administration combined with that of nicotine in rats. Cross-dependence is reported between nicotine and alcohol in alcoholic subjects. Recent studies are cited to suggest that other than cholinergic binding sites in the brain are responsible for the effects of nicotine on the central nervous system.

84–0247. Mangan, G. L. **The Effects of Cigarette Smoking on Verbal Learning and Retention.** *Journal of General Psychology* 108(Second Half): 203-210, 1983.

Cigarettes with either .7 mg or 1.3 mg nicotine level significantly improved retention in paired-associate learning, with task difficulty apparently having little relevance. The effects of smoking a low (.7 mg) and a middle (1.3 mg) nicotine yield cigarette on paired-associate learning and retention under conditions of high and low intralist interference, and on serial learning and retention, were examined in groups of male undergraduate smokers (n = 24). The interaction between nicotine level and task difficulty in paired-associate learning was marginally significant. The 1.3 mg nicotine dose impeded learning under low interference conditions, but facilitated learning of high interference sets. Serial learning data suggested that the effect is shown on long-term, rather than short-term memory. (Auth. Abs. Mod.)

84–0248. McMorrow, M. J.; Foxx, R. M. **Nicotine's Role in Smoking: An Analysis of Nicotine Regulation.** *Psychological Bulletin* 93(2): 302-327, March 1983.

Nicotine regulation refers to a smoker's maintenance of a characteristic level of nicotine in the body, and implies that changes in smoking behavior (i.e., compensation) will accompany either increases or decreases in nicotine availability. This review notes that most research on regulation has been inconclusive due to (1) a failure to distinguish between regulation and compensation; (2) the use of indirect rather than direct measures of nicotine exposure; and (3) methodological problems. An attempt is made to (1) clarify definitional ambiguities, (2) present a classification strategy that differentiates the indirect and direct evidence for regulation, (3) quantify the direct evidence, (4) discuss measurement and methodological problems, and (5) suggest future research. (Auth. Abs. Mod.)

84–0249. Mitchell, D. R. **A Comparison of High School Students' Smoking Behaviours in 1968 and 1981.** *New Zealand Medical Journal* 96(734): 534-536, July 13, 1983.

A questionnaire survey conducted among students in three Gisborne, New Zealand, high schools in 1968 (n=514) and 1981 (n=583) showed a shift toward increased consumption among females and declining prevalence among males for both Maori and European students. The proportion of Maori females reporting frequent smoking rose from 12.7 percent in 1968 to 39.3 percent in 1981; corresponding figures for European females were 5.6 and 29.3 percent, respectively. Conversely, the proportion of Maori males who were never smokers rose from 36.5 percent in 1968 to 50.5 percent in 1981; corresponding figures for European males were 37.6 and 56.6 percent, respectively. While socioeconomic status and smoking prevalence were not related to either ethnic group in 1968, survey results for 1981 showed statistically significant differences between high and low socioeconomic groups among European students, but not for Maori students. European students from lower status homes reported higher levels of smoking. The 1981 sample showed a statistically significant tendency for higher consumption among Maori students with high cultural identity (31 percent frequent smokers), compared with those with low cultural identity (14 percent frequent smokers). Of students reporting frequent smoking, 39.5 percent also reported frequent alcohol consumption. Only 5.6 percent of never smokers reported frequent alcohol consumption.

84–0250. Mitchell, D R. **Alcohol Consumption of High School Students in Relation to Ethnicity, Socioeconomic Status, and Sex.** *New Zealand Medical Journal* 96(736): 572-575, July 27, 1983.

Results of a 1981 survey of 554 fifth form students (218 Maoris, 107 males and 111 females; 336 Europeans, 152 males and 184 females) in three New Zealand high schools showed a 90 percent probability that those who frequently consumed alcohol were also smokers. Of those students who often consumed alcohol, 54.1 percent often smoked while 11.1 percent rarely or never smoked. However, students who never consumed alcohol were evenly distributed across all smoking categories. Percentage frequencies of students' alcohol consumption by smoking behavior are provided. Overall results showed a shift toward higher reported levels of alcohol consumption among females (from 18.3 percent in 1968 to 35.3 percent in 1981). Male levels showed a slight (not significant) decline. Sex differences applied to both Maori and European students over the survey period.

84–0251. Morbidity and Mortality Weekly Report. **Behavioral Risk Factor Prevalence Surveys — United States, Second Quarter 1982.** *Morbidity and Mortality Weekly Report* 32(28): 370-372, July 22, 1983.

Survey data showed that the proportion of adults reporting cigarette smoking ranged from a low of 23.4 percent in Kansas to a high of 37.1 percent in Kentucky and North Carolina. During the second quarter of 1982, eight States (Delaware, Indiana, Kansas, Kentucky, Montana, New Jersey, North Carolina, and Ohio) and the District of Columbia conducted the prevalence study of major behavioral risk factors among adults through random telephone surveys. A standard questionnaire was used in six States and the District of Columbia. Self-reported data were adjusted for sex, and weighted according to the respondents' probability of selection. Nationally, the most recent report (1980) showed that the level of cigarette smoking was 32.4 percent. Other risk factors surveyed were obesity, sedentary lifestyle, uncontrolled hypertension, acute heavy drinking, chronic heavy drinking, drinking and driving, and lack of seatbelt use. Data are listed by sex for four age groups: 18 to 34, 35 to 54, ≥ 54 years, and for all ages.

84–0252. Muramatsu, S.; Muramatsu, T.; Weber, A. **A Survey on Attitudes Towards Passive Smoking Among Schoolchildren and Students in Switzerland.** *Sozial- und Praventivmedizin* 28(2): 82-84, 1983.

Questionnaire data on attitudes and symptoms related to passive smoking were collected from 183 13-year-olds, 203 16-year-olds, and 367 23-year-old students in Zurich. Significantly more of the younger children (81 percent) than older children (70 percent) and students (70 percent) disliked passive smoking, and significantly more nonsmokers than smokers disliked passive smoking. The subjects whose parents smoked disliked passive smoking less than those whose parents did not smoke. A total of 80 percent of the students (64 percent of the older children and 82 percent of the younger children) complained of one or more kinds of irritation due to passive smoking; the most common was eye irritation, followed by cough, and nose irritation. More nonsmokers complained of irritation than smokers, but no significant differences were noted between girls and boys in their reactions to passive smoking. (Auth. Abs. Mod.)

84–0253. Nichols, J. R. **The Homeostatic Reflex and Addictive Drugs.** *Neurobehavioral Toxicology and Teratology* 5(2): 237-240, March-April 1983.

Tobacco is discussed in terms of its addictive power, as are other drugs including alcohol. The homeostatic reflex, a universal compelling mechanism controlling actions, changes the nervous system so that aversive stimuli from a homeostatic (internal stability) imbalance are associated with a response that turns them off. Addictive drugs compel actions by invoking this mechanism to turn off biological drives. Tobacco reduces anxiety, hunger, and sex drives; smokers smoke more under stress and eat less than nonsmokers. But withdrawal causes a rebound effect: increased distress that can be relieved by readministering the drug. Drugs also create a multidrive reflex, turning off many drives simultaneously, whereas normal actions usually turn off only one. Thus, the drug-taking action results in habit reinforcement, creating an even stronger reflex action. Our survival mechanism of reflex actions has many benefits, making us do things that are good for us. However, it also compels senseless actions like clinical compulsions and the addictions.

84–0254. Oleinikov, S. P.; Chazova, L. V.; Glazunov, I. S.; Deev, A. D.; Baubinene, A. V.; Prokhorskas, R. P.; Domarkene, S. B. **Kureniye i Nekotoryye Sotsial'no-Demograficheskiye Kharakteristiki (Dannyye Kooperativnogo Issledovaniya v Moskve i Kaunase). [Tobacco Smoking and Some Socio-demographic Characteristics (Data of Cooperative Studies in Moscow and Kaunas).]** *Terapevticheskii Arkhiv* 55(1): 57-61, 1983, Russian.

Examination of randomized samples of men 40 to 59 years of age in Moscow and Kaunas demonstrated the relationship of smoking with age, educational level, and occupation. With advancing age, the number of tobacco smokers decreased by 10.7 percent and 8.7 percent in Moscow and Kaunas, respectively. Among the subjects with a higher educational level, the percentage of tobacco smokers was lower compared with men with secondary, incomplete secondary, or primary education. The percentage of tobacco smokers among intellectuals was lower than that among the personnel with medium-technical education and workers. All the features of tobacco smoking distribution, depending on the sociodemographic characteristics studied, were statistically significant. (Auth. Abs.)

84–0255. OPCS Monitor. **Cigarette Smoking: 1972 to 1982.** *OPCS Monitor* 83(3): 1-8, July 5, 1983.

Data for Great Britain show that in 1982, 38 percent of men and 33 percent of women were smokers, a substantial decline from 1972 (14 percent drop in males, and an overall reduction of about 20 percent among women). Cigarette consumption statistics for Great Britain, collected in the General Household Survey (GHS), are reported for alternate years from 1972 through 1982 (except 1974). Data on prevalence of smoking and average weekly consumption are given in tables by age, sex, and socioeconomic group. Overall weekly consumption among male smokers was lower in 1982 than in 1980. Declines in smoking prevalence occurred in all age groups except women aged 20 to 24, and in all socioeconomic groups, although weekly consumption among smoking males in the professional—managerial group increased by 10 percent.

84–0256. Paty, J.; Brenot, P.; Deliac, P.; Dogui, M. **Un Element d'Evaluation de la Dependance Tabagique. Naloxone et V.C.N. [Contribution to Evaluation of Smoking Dependence. Naloxone and Contingent Negative Variation (C.N.V.).]** In: Molimard, R. (Editor). *1ere Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 135-144, French.

While contingent negative variation (CNV) of brain potential in response to smoking and naloxone does not fully explain the mechanism of human smoking dependence, results indicate a relationship between morphine receptors and smoking. Using protocols developed in the Laboratory of Experimental Medicine of Bordeaux University, CNV recordings were made in (1) 25 subjects before a 5-day behavior modification smoking withdrawal program, and 1 year later under various conditions; (2) five regular smokers of one pack/day and five nonsmokers of similar age, before and after smoking four cigarettes, and, subsequently, each 5 minutes after i.v. injection of 5 cc of naloxone; and (3) undetermined number of subjects before sexual intercourse, 2 hours later, and each 5 minutes after subsequent injections of naloxone. Indications are that (1) CNV amplitude increase after smoking withdrawal cure is of neurobiological nature; (2) CNV amplitude in smokers decreases after smoking but reappears after naloxone injection, decreasing again after 15 minutes (nonsmokers experience a variety of reactions); and (3) CNV amplitude variations after intercourse were similar to those after smoking. Physiological and psychological significance of observed reactions to smoking and naloxone are unclear.

84–0257. Pomerleau, O. F.; Fertig, J. B.; Shanahan, S. O. **Nicotine Dependence in Cigarette Smoking: An Empirically-Based, Multivariate Model.** *Pharmacology Biochemistry and Behavior* 19(2): 291-299, August 1983.

A coherent model of cigarette smoking was developed by examining the responses of 27 habitual smokers on several different measures of nicotine dependence. In the laboratory, subjects filled out questionnaires before and after smoking research cigarettes differing in nicotine content. Plasma cotinine was used to estimate nicotine intake from usual brand cigarettes smoked outside the laboratory. Subjects in the high-cotinine quartile (heavy smokers) were found to be consistently more nicotine-dependent than subjects in the low-cotinine quartile (light smokers). For all subjects, the six measures of nicotine dependence that exhibited significant correlations with plasma cotinine accounted for about half of the cotinine variance in a multivariate, linear regression model. Multivariate approaches provide additional tools for assessing biobehavioral mechanisms in substance abuse and may lead to the development of more comprehensive and sufficient explanations of smoking than are currently available. (Auth. Abs. Mod.)

84–0258. Salmons, P. H.; Sims, A. **Smoking Profiles of Patients Admitted for Neurosis. (Letter).** *British Journal of Psychiatry* 141: 103, July 1982.

According to a previous letter (*British Journal of Psychiatry* 140: 102, January 1982) age at starting to smoke differed between neurotics and controls only because of the disproportionately small number of neurotics in the older group. The authors of the original article respond, noting that only 49.7 percent of males and 23.6 percent of females in the general population had begun smoking by age 17. Among the neurotics, however, 63.7 percent of males and 33.3 percent of females were smokers at age 17. This leaves fewer neurotics to begin smoking at a later age. Smoking is not claimed as a panacea for neuroticism, though neuroticism seems to be a contributing factor in maintenance of the smoking habit. See also 82-1099.

84–0259. Shaffer, H.; Beck, J. C.; Boothroyd, P. **The Primary Prevention of Smoking Onset: An Inoculation Approach.** *Journal of Psychoactive Drugs* 15(3): 177-184, July-September 1983.

A comprehensive smoking intervention and prevention program (inoculation approach) is described that effectively prevented the onset of smoking and unexpectedly reduced smoking levels in 114 seventh grade students (60 boys and 54 girls). The program utilized three basic phases: (1) cognitive preparation, (2) skill acquisition, and (3) rehearsal of situational responses. Smoking as an emotional and behavioral response to situational demands requiring problem-solving strategies was emphasized. Prevention elements were designed to enable students to recognize the internal and external factors that could influence their desire to smoke, and to teach coping skills that would help them manage their cognitive and behavioral responses. Of students (n = 75) in the program, 8.9 percent had been daily smokers. Following the program, only 5.1 percent reported daily smoking. Among the 35 control students the percentage of daily smokers increased from 8.6 percent to 9.7 percent over the same period. For other smoking categories, 5.0 percent quit entirely, and percentages of occasional smokers (weekly and monthly) were reduced. The manual prepared for this program can be used by others to run similar projects.

84–0260. Shapiro, L. R.; Samuels, S.; Breslow, L.; Camacho, T. **Patterns of Vitamin C Intake From Food and Supplements: Survey of an Adult Population in Alameda County, California.** *American Journal of Public Health* 73(7): 773-778, July 1983.

Smoking was inversely related to vitamin C intake among 3,119 adults (1,044 smokers, 642 ex-smokers, 1,295 never smokers) of Alameda County, California. The vitamin C intake of nearly 80 percent of respondents met or exceeded recommended daily allowances: only 14 percent or fewer appeared to have inadequate vitamin C intake. Respondents who smoked cigarettes or did not eat breakfast had lower vitamin C intakes. Vitamin C supplements were taken daily by 29 percent, occasionally by 21 percent. Most respondents obtained 100 mg and or less of vitamin C daily, while 2 percent consumed more than 2,000 mg. Proportionately, more women than men took vitamin C pills daily, and men aged 16 to 44 were the least likely to consume either vitamin C foods or pills. Persons 45 years and older tended to take vitamin C pills daily, while younger persons took them only occasionally. Overall, people in poor health consumed more vitamin C than those in good health. (Auth. Abs. Mod.)

84–0261. Sherman, S. J.; Presson, C. C.; Chassin, L.; Corty, E.; Olshavsky, R. **The False Consensus Effect in Estimates of Smoking Prevalence. Underlying Mechanisms.** *Personality and Social Psychology Bulletin* 9(2): 197-207, June 1983.

There was a consistent false consensus effect (FCE) for estimates of smoking prevalence by adolescents (n = 5,291), but not for estimates by adults (n = 122). Adolescents (grades 6 through 12) who smoked (12.5 percent in midwestern subjects, 15.5 percent in southwestern subjects) estimated significantly higher numbers of smokers than those who did not smoke. Adult smokers and nonsmokers did not differ in their estimates of smoking prevalence among men, women, and 14- to 16-year-old boys and girls. The role of selective exposure, motivational distortion, causal attributions, and behavioral conformity were investigated as possible mechanisms underlying the FCE. Results show little support for the attributional or conformity interpretations of the FCE in adolescents' estimates, but support both the selective exposure and motivational theories. The selected exposure approach views the FCE as the outcome of a calculated decisionmaking process. The motivational interpretation sees the FCE as an egocentric bias that has its roots in needs for self-enhancement and self-validation rather than in cognitive judgmental principles. It is noted that both adults and adolescents greatly overestimated the actual smoking rates of the populations in question.

84–0262. Sjoberg, L. **Value Change and Relapse Following a Decision to Quit or Reduce Smoking.** *Scandinavian Journal of Psychology* 24(2): 137-148, 1983.

Data from 23 smokers (16 men, 7 women, mean age 27.3 years) who tried to quit or reduce smoking support the theory that values and beliefs are indicative of stereotypical, biased thinking during an attempt to quit smoking. An earlier model (Ainslie) had suggested that values change subsequent to a decision, and the value of a smaller, immediately available reward increases at a faster rate than that of a later reward. The rank order between the two rewards may therefore become reversed, leading to impulsive behavior. Neither model was successful in accounting for relapses during the 1-week trial, possibly because action is hard to predict from beliefs and values. However, answers to daily questionnaires, which rated 35 selected events and states, showed that those smokers who perceived themselves as persistent also had a stronger, and more consistent, trend toward change of values, becoming more negative to smoking as the week passed. Relapse may be the result of craving, but also loss of control due to extraneous events of an emotionally provocative nature, the use of drugs or alcohol, or other factors.

84–0263. Smith, G. M.; Schwerin, F. T.; Stubblefield, F. S.; Fogg, C. P. **Licit and Illicit Substance Use by Adolescents:**

Psychosocial Predisposition and Escalatory Outcome. *Contemporary Drug Problems* 11(1): 75-100, Spring 1982.

A 400-item questionnaire designed to evaluate personal competence and social responsibility was administered to 1,474 students (7th through 9th grades) in 1969, and again 3 years later (when the students were in grades 10 through 12). Peer rating data and tobacco, alcohol, and cannabis consumption information were also collected. The students who had begun using licit substances (cigarettes and alcohol) at the time of the second survey differed significantly from other students in psychosocial predisposition scores. The six psychosocial variables were (1) obedient (self-report); (2) obedient (peer rating); (3) sociable (peer rating); (4) grade point average; (5) negative cigarette attitudes; and (6) psychosocial composite. Overall, the data demonstrate that personality and other psychosocial variables are related to and predictive of both licit and illicit substance use. It is also shown that the interactive process begins early in preadolescence, influenced by (1) personality formation of individual children; (2) reinforcement by like-minded children; (3) differences among groups regarding attitudes toward, and the use of, substances; and (4) differential availability of substances to such groups. The strength of the psychosocial predisposition is greater among adolescents who will escalate to illicit use (cannabis) than among those who will stop short of illicit use (alcohol and cigarettes), and greatest in those who will become frequent users of illicit substances.

84–0264. Spencer, J. K. **Nurses and Cigarette Smoking: A Literature Review.** *Journal of Advanced Nursing* 8(3): 237-244, May 1983.

A brief literature review (48 references) critically examines assumptions leading to conclusions that nurses are heavy smokers. One study in the United Kingdom reported that 48 percent of nurses smoked, but failed to distinguish between professionally trained nurses and those performing nursing tasks. The choice of comparison groups is questioned, stating that male, professional, high-status physicians provide an invalid comparison group. Better comparisons might be made with other occupational groups of women, stratified by socioeconomic status. Additionally, surveys have failed to consider the distinction between nurses who were smokers before they became nurses and those who began smoking during training or after their nursing careers began. The influence of nurses' smoking on health education campaigns, particularly among hospital patients, needs redefining. It is stated that if health education is defined as the providing of information, personal behavior should not be a factor in choosing the educator.

84–0265. Suta, B. E.; Thompson, C. R. **Smoking Patterns of Motor Vehicle Industry Workers and Their Impact on Lung Cancer Mortality Rates.** *Journal of Occupational Medicine* 25(9): 661-667, September 1983.

Differences in cigarette consumption account for 35 to 80 percent of the excess in lung cancer mortality among white male automotive workers. Recent data have shown that blue-collar automotive workers have an excess of approximately 30 percent in lung cancer deaths over a comparable sample of the general population. The smoking habits of automotive workers were compared with those of the general population to assess the extent to which any smoking habit differences might account for the excess in lung cancer mortality. Results show that, when smoking differences are considered, the lung cancer proportionate mortality ratio for white male automotive workers declines from 1.3 to approximately 1.1. (Auth. Abs. Mod.)

84–0266. Suter, T. W.; Battig, K. **Vegetative Effects of STROOP-Test Solving and Cigarette Smoking.** *Activitas Nervosa Superior* 2(Supplement 3): 264-267, 1982.

Psychophysiological reactivity to task solving showed some tendency to be influenced by smoking in an investigation of the interaction between smoking-induced and task-induced physiological arousal in 28 male smokers. After a training session, subjects performed STROOP tasks before and after smoking either a 0.2 mg nicotine or a 1.2 mg nicotine cigarette. Physiological arousal was monitored by measuring EKG, EMG, EEG, finger pulse volume, respiration, and skin conductance. A control group ($n = 16$) was monitored physiologically after smoking but did not perform STROOP tests. The control group exhibited significant elevation in heart rate ($p = 0.001$) and decreased pulse amplitude ($p = 0.001$); no significant changes were observed in the test subjects. In general, effects of smoking were considerably more pronounced in the control group than in the task-solving test group.

84–0267. Tucker, L. A. **Cigarette Smoking Intentions and Obesity Among High School Males.** *Psychological Reports* 52: 530, April 1983.

High school boys who are self-perceived endomorphs, or relatively obese, have significantly stronger intentions of smoking cigarettes than their counterparts. Data from 400 high school males (51 endomorphs, 189 mesomorphs, 157 ectomorphs) were analyzed to determine if smoking intentions differed significantly. Relatively obese subjects scored significantly higher on the measure of smoking intentions compared with moderately built subjects and lean males. This association may be mediated by several factors: self-esteem, anxiety, and physical fitness. In addition, since the behavior of students is functionally related to the general social positions they occupy, the significant cigarette smoking intentions—obesity association found may be a function of peer acceptance and social status.

84–0268. Warner, K. E. **Reactions to Perceived Risk: Changes in the Behavior of Cigarette Smokers.** In: Covello, V. T.; Flamm, W. G.; Rodricks, J. V.; Tardiff, R. G. (Editors). *The*

Analysis of Actual Versus Perceived Risks. New York, Plenum Publishing Corporation, 1983, pp. 183-201.

While smoking remains a persistent national health problem, behavioral changes reflecting perceived smoking-related personal health risk and social undesirability have increased steadily since publication of the original Surgeon General's report in 1964. Per capita estimates for consumption in the absence of the antismoking campaign were 40 percent greater than actual consumption in 1978. Among males, self-reported smoking rates have decreased persistently and significantly and, although smoking prevalence among females rose through the 1960s, trends show gradually declining rates through the 1970s. Data for the combined influence of these trends show that smoking rates for males more than doubled the rates for females prior to the 1964 report, but that the differential declined to one-third by 1975. Surveys for 1968 through 1979 show that overall teenage rates are declining, while prevalence among teenage females has reached parity with that of males. The gradual drop in per capita consumption actually reflects a more rapid decrease in the proportion of smokers, moderated by an increase in quantity consumed by the average continuing smoker (partially attributed to nicotine compensation among those shifting toward low-yield brands). Health education in the 1980s should emphasize (1) the risk of subtle changes in smoking behavior among low-tar and low-nicotine smokers (i.e., increased consumption, deeper inhalation, covering filtration holes, smoking further down the cigarette, more frequent puffing); and (2) the possible health hazards of additives used to enhance flavor and improve burning, several of which are known or suspected carcinogens. Provided are data for self-reported smoking rates, by year, for males and females in the United States, among (1) adults from 1955 to 1975, and (2) teenagers, by age, from 1968 to 1979; total adult per capita consumption, by year, from 1960 to 1980; per capita consumption, actual and predicted in the absence of the antismoking campaign through 1980; filter tip share of the cigarette market, 1949 to 1978; sales weighted average tar and nicotine/cigarette, 1954 to 1978; and actual and reported cigarette consumption, 1964 to 1975. It is stated that survey respondents significantly underreport consumption, and that the amount of underreporting has increased since the original Surgeon General's report.

84–0269. Yanagita, T.; Ando, K.; Kato, S.; Takada, K. **Psychopharmacological Studies on Nicotine and Tobacco Smoking in Rhesus Monkeys.** *Psychopharmacology Bulletin* 19(3): 409-412, 1983.

Results of a differential reinforcement of low rate (DRL) schedule and discrimination experiments in rhesus monkeys suggest that nicotine partially shares the psychopharmacological properties of *d*-amphetamine, morphine, and diazepam. Areas studied over the last decade include intravenous self-administration of nicotine, cigarette smoking behavior, behavioral effects under a DRL schedule, and discriminative stimulus properties of nicotine. Intravenous self-administration experiments (cross self-administration, continuous self-administration, progressive test ratio) have yielded equivocal results, possibly attributable to procedural differences and the relatively low reinforcing properties of nicotine. Monkeys never overdosed nicotine to the point of acute toxic effects, perhaps due to a nicotine-related self-limiting factor (i.e., dysphoric effects at overdose). During intensive cigarette-smoking experiments in monkeys, none successfully maintained long-term smoking behavior without combining additional reinforcers with smoking. While a test with nicotine-free cigarettes showed that smoking behavior is primarily reinforced by nicotine, other data indicate that behavior is not necessarily maintained by nicotine.

84–0270. Zeiner-Henriksen, T. **Landsforeningen Mot Kreft's Undersokelse av Rokevaner Viser: Faerre Tenaringer Begynner a Roke, Kvinner "pa Stedet Hvil," Fortsatt Hoyt Rokeniva Blant Voksne Menn, Storst Nedgangi Roking Blant dem Med Lengst Skolegang. [National Cancer Institute's Study of Smoking Habits Reveals: Fewer Teenagers Are Beginning to Smoke, Women "Are at Ease," High Smoking Levels Continue Among Adult Men, Highest Reduction in Smoking Among Those With the Most Education.]** *Mot Kreft* 33(1): 16-17, 1983, Norwegian.

According to the Institute for Cancer Research, Norwegian Radium Hospital, a slight reduction in smoking among men, but not among women, was noted between 1980 and 1982. A drop was also seen among both males and females in the youngest age group. Since 1972, smoking among the least educated males has fallen from 57 to 49 percent, and from 50 to 33 percent among highly educated males. In women, smoking fell only 7 percent in the most educated and 3 percent in the least educated. Among the smokers, however, consumption has increased 9 percent in men and 4 to 5 percent in women since 1980. Average consumption is now 14.8 cigarettes/day for men and 12.5/day for women (highest in men aged 25 to 29, and in women aged 30 to 39 years). It is noted that the age of smoking onset has also increased since 1980 (from 15.2 to 16.5 years in men, and from 15.2 to 15.9 years in women).

See also, **84–0091, 84–0183, 84–0333**

SMOKING PREVENTION AND INTERVENTION

84–0271. Bauman, K. E.; Bryan, E. S.; Dent, C. W.; Koch, G. G. **The Influence of Observing Carbon Monoxide Level on Cigarette Smoking by Public Prenatal Patients.** *American Journal of Public Health* 73(9): 1089-1091, September 1983.

Observation of their own alveolar carbon monoxide (CO) level had little or no influence on smoking habits among 170 pregnant women (47 percent smokers) in Guilford County, North Carolina. All subjects were read a 135-word script describing the relationship between smoking, CO, and the health effects of smoking during pregnancy. Women in the experimental group provided a breath specimen and observed machine needle readings (in ppm) of their own and others' CO levels. Six weeks after the intervention, no significant difference in behavior was evident between the experimental and control groups, although when repeat analysis included 12 socioeconomic and demographic variables in the model, mean CO level was lower in the experimental (14.6 ppm, $p<0.04$) than in the control group (20.1 ppm).

84–0272. Best, J. A.; Bloch, M. **Compliance in the Control of Cigarette Smoking.** In: Haynes, R. B.; Taylor, D. W.; Sackett, D. L. (Editors). *Compliance in Health Care.* Baltimore, Johns Hopkins University Press, 1979, pp. 202-222, 488-497.

The concept of compliance in smoking cessation (regimen adherence vs. outcome) is explored, and the techniques and success rates of five basic smoking modification strategies are discussed. These include group therapy, education and attitude change, pharmacological therapy, hypnosis, and behavior modification (aversion, stimulus control, self-monitoring, anxiety management, response substitution, contingency management, social support, and maintenance procedures). A sample multifaceted behavioral modification program is outlined. Suggested research priorities include improving program effectiveness, subject compliance, and community services.

84–0273. Bostock, Y.; Hillhouse, A. J.; Inglis, V. **Pharmacists and Health Education: An Evaluation of a Stop-Smoking Project Involving Pharmacists.** *Health Education Journal* 42(1): 20-23, 1983.

Of 400 retail outlet pharmacists invited to participate in an antismoking campaign, 150 (20 percent smokers) responded and received stop-smoking materials including window display posters, "shelf wobblers," smoking cessation leaflets, and "Ask me" badges for pharmacists' assistants (60 percent smokers). Telephone interviews indicated that two-thirds of the pharmacists used the "shelf wobblers"; 27 percent did not use the "Ask me" badges; 54 percent distributed over 100 leaflets carrying the message "Want to stop smoking? Ask here"; 58 percent had more than 26 customer requests for smoking cessation advice and assistance; and three-fourths had discussed smoking cessation with customers and believed the project was very effective. Ninety-five percent responded that they would participate in future projects. Face-to-face interviews indicated that 15 percent of nonparticipants chose to remain uninvolved because they were smokers. While over 90 percent of participants, nonparticipants, and controls (noninvolved pharmacies) dispensed advice several times daily, only 63 percent of nonparticipants, compared with 91 percent of participants and 85 percent of controls, mentioned smoking (statistically significant). It is concluded that pharmacists have a broader role in health education and health care than previously realized, due to their active dispensing of health advice and direct, influential access to the public.

84–0274. Catford, J. C.; Nutbeam, D. **Promoting Health, Preventing Disease: What Should the NHS Be Doing Now?** *Health Education Journal* 42(1): 7-12, 1983.

Considerable mortality and disability could be avoided through prevention of smoking, and action oriented toward hypertension, nutrition and exercise, accidents, prenatal care, immunization, and child health screening. However, National Health Service barriers to health promotion and disease prevention have included deficiencies in the areas of defined responsibility, coordinated work force, direction, information, involvement, professionalism, imagination, and resources. Recommended are the "5 I's": identify, involve, inform, invest, and innovate.

84–0275. Dismuke, S. E.; Miller, S. T. **Why Not Share the Secrets of Good Health?** *Journal of the American Medical Association* 249(23): 3181-3183, June 17, 1983.

Because of contact with people during times of crisis, the physician is in a unique position to help patients modify long-term adverse health behaviors such as smoking and obesity. Smoking can be linked to respiratory conditions that commonly prompt office visits. The patient can be counseled by the physician or referred to other programs designed to effectively change unhealthy habits. However, while physicians smoke much less than the general population, and many who formerly smoked have quit, many are reluctant or ineffective in communicating the health risks of smoking to their patients. Medical schools, which generally do not teach the skills necessary for the practice of health promotion, must share the blame. Some of the newer health care organizations, hospitals, and private corporations are responding to the public's demand for prevention and health enhancement, but physicians should be aware of the impact they can make on a patient's behavior. Studies have shown that physicians who emphasize smoking cessation during routine office visits can be effective in getting their patients to stop smoking. It is time for physicians to share their secrets for good health and become active proponents of health promotion.

84–0276. Health and Welfare Canada. **Canadian Initiatives in Smoking and Health.** Health and Welfare Canada, Publication No. H39-67/1983, 1983, 130 pp.

The Department of National Health and Welfare describes how Canada proceeded against the problem of smoking after research had established that smoking is dangerous to health. Since 1963, public information and education programs have helped to create a social climate where nonsmoking is an attractive and healthy personal choice. Between 1966 and 1981, the proportion of adult Canadian men who smoke regularly dropped from 54 percent to 36 percent; for adult women, the decline was from 32 percent to 29 percent. Long-term programs are aimed at creating an entire generation of nonsmokers. The early efforts, initial successes, formation of the Canadian Council on Smoking and Health (1974), the growing awareness that smoking is a lifestyle as well as a medical problem, and recent Federal and provincial programs are discussed. A major new program, "Generation of Nonsmokers," will consist of (1) high-profile advertising; (2) special materials for children and parents; and (3) efforts to secure participation of interested groups. While the Generation program hopes to prevent smoking, the "Time to Quit" program will focus on current smokers, utilizing a multimedia, community-based approach aimed at smokers between the ages of 25 and 45.

84–0277. Hill, D. **The Role of the Doctor in Patient Smoking Cessation.** *Cancer Forum* 7(1): 5-8, March 1983.

Physicians need to be convinced that smoking cessation efforts with patients are worthwhile, and, that given the relative expense and time, a 5 percent cessation rate is good. A general practitioner giving 2 minutes of firm cessation counseling and a how-to-quit leaflet could produce 25 ex-smokers/year. When physician pressure is insufficient to produce cessation, nicotine chewing gum may be utilized. Nicotine gum is pharmacologically effective, reduces withdrawal symptoms, and is easily prescribed. The stereotyped role of a doctor may actually make both the physician and patient more comfortable during cessation counseling. However, doctors should not be encouraged to suggest using more drugs, since such a practice could lead to the acceptance of smoking as a habit only a doctor can "cure." The GUS kit (give up smoking) for general practitioners, developed by Action on Smoking and Health and the Health Education Council, is described.

84–0278. Lee, K. **A Designing Health Education Program. Cigarette Smoking and Health for Junior High School.** *Korean Nurse* 119: 47-64, January-February 1983.

An educational program on smoking and health is provided for use in Korean junior high schools. The program's goal is to help students to understand the effects of cigarette smoking and to prevent smoking initiation. Five lesson plans are given: immediate effects of tobacco, potential effects of smoking, nicotine dependence, comparison of beneficial and detrimental effects of smoking (given as a debate format), and creation of an antismoking campaign. An examination covering the five lessons is included. Since over half the men in Korea smoke, it is felt that the school nurse at the junior high school level can make a valuable contribution to health by using this type of program before smoking habits are established.

84–0279. Leroux, R. S.; Miller, M. E. **Electronic Media-Based Smoking Cessation Clinic, In the USA.** *International Journal of Health Education* 2(1): 23-35, March 1983.

A smoking cessation program using radio and television was provided to residents of Denver, Colorado. Two weeks of promotion, which provided a Quit Line telephone number and 1 week of daytime programing, were given. Respondents who called the Quit Line were sent a smoking kit, and two followups (at 2 and 3 months) were made. Packets were requested by 3,806 respondents. Of these, 544 smokers were interviewed (the Media Clinic Group or MCG) and a comparison group (n=404, 326 smokers) was chosen from the area. The MCG was older, smoked more, was more highly educated, and was more concerned about health problems than the comparison group. After the program, changes in smoking behavior were reported by 71 percent of the MCG and by 12 percent of the comparison group. While success of the program is difficult to determine, 33 percent of the MCG reported that they were very likely to quit smoking. The total cost of the program was less than 2 dollars/person.

84–0280. Luepker, R. V.; Johnson, C. A.; Murray, D. M.; Pechacek, T. F. **Prevention of Cigarette Smoking: Three-Year Follow-Up of an Education Program for Youth.** *Journal of Behavioral Medicine* 6(1): 53-62, March 1983.

Data from a 3-year followup of students who had participated in an innovative approach to smoking prevention show that smoking behavior appears significantly inhibited by a peer-led curriculum and that the effect is retained for several years. Initially, 1,081 seventh grade students were involved in separate programs at three Minnesota schools: (1) monitoring control (questionnaire on usage and saliva thiocyanate test); (2) social pressures curriculum/monitoring; and (3) social pressures curriculum/monitoring/presentation by peer leaders. The curriculum emphasized the short-term influences that affect smoking in youth, particularly social and peer pressures. In the school that received this curriculum from like-aged peer leaders, the incidence of smoking remained low compared with incidence in the control school. Those who did smoke in the treatment school consumed significantly fewer cigarettes. In the school where the curriculum was taught by adults, smoking rates were initially lower but rose in later years, ultimately differing little from those in the control school. Reported smoking behavior was confirmed by saliva thiocyanate measurement in all students. Residual positive effects of the program are observed for 1 year in the adult-led groups and several years in the peer-led group. However, it also appears that a second program, timed for ninth or tenth grade, may be necessary to sustain long-term reductions in smoking. (Auth. Abs. Mod.)

84–0281. Neukirch, F.; Cooreman, J. **Les Resultats des Tests de la Fonction Pulmonaire, et la Mise en Evidence de Symptomes Respiratoires Lies au Tabac Peuvent-ils Etre Utilises Pour Dissuader les Adolescents de Fumer? [Can Results of the Pulmonary Function Tests and the Evidence of the Smoking-Related Respiratory Symptoms Be Utilized to Dissuade Teenagers From Smoking?]** In: Molimard, R. (Editor). *1^ere^ Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 130-134, French.

Questionnaires about respiratory symptoms and smoking habits were administered to (1) 29,138 students aged 14 to 19 in 1976; (2) 7,963 students aged 14 to 18 in 1979; and (3) 2,269 students aged 10 to 19 in 1978-1979. Respiratory function was measured simultaneously in 971 of the students and 2 years later in 236 of the same students. Data showed an instantaneous respiration rate reduction of at least 30 percent of the theoretical value. In all three surveys, a very significant correlation was established between smoking and respiratory symptoms. For the third survey, incidence of symptoms was higher in female than in male smokers. Presentation of these results to the students attracted a great deal of attention and had a greater impact than fear of a future chronic illness. The smoking reduction noted in Paris students 2 years after the initial survey was significantly correlated with the evidence of respiratory symptoms in the first year. For smoking girls (but not boys), the instant respiration rate was significantly reduced and forced expiratory volume in 1 sec was inversely related to the number of cigarettes smoked. It is concluded that information received at the elementary school level is the only significant antismoking measure.

84–0282. Said, G.; Mann, I.; Nalpas, J. **Essai de Prevention Tabagique Dans les Ecoles et Lycees des Hauts de Seine. [An Attempt at Smoking Prevention in the Schools and Lyceums of the Hauts de Seine.]** In: Molimard, R. (Editor). *1^ere^ Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 123-129, French.

In an effort to prevent smoking among youth, the Departmental Committee of the French National Cancer League has been sponsoring 60 to 80 information meetings each year for 9- to 13-year-old students of the elementary and secondary schools. The purpose of the meetings is to stimulate reasoned behavior and begin antismoking education at the earliest possible age to counterbalance the social pressures to smoke. An attempt is made to explain to students the harmful effects of smoking to both themselves and their environment, to describe the manipulation of society by advertisements, and to help students appreciate a healthy lifestyle. Followup showed that none of the students who attended the information meetings at an early age had become regular smokers in their last year of school, while 45 percent of the other last-year students had become smokers. It is concluded that the described preventive action with young children is efficient.

84–0283. Totten, M. A.; Sherin, K.; Wechsler, H. **Physician's Role in Health Promotion. (Letters).** *New England Journal of Medicine* 308(23): 1424-1425, June 9, 1983.

It is suggested that an earlier report (*New England Journal of Medicine* 308(2): 97-100, January 13, 1983), overstated the interest of physicians in health promotion, since only 27 percent of those surveyed routinely gathered information from patients on smoking, alcohol consumption, drugs, stress, exercise, and diet. Many of the present barriers to more active participation are the result of the development of medicine as a highly scientific, technologic, and curative profession, leaving less time for patient education. An alternative approach would be for physicians to identify patients' health risks and provide guidance for other health-care professionals who are more adept at providing the necessary services. In reply, the original author agrees that physicians cannot provide all services for all people, but feels they are responsible for making sure patients receive health education, either in their offices, or by referral to appropriate sources. To do this, physicians need help in obtaining information about available resources within the community. The low level of importance attached to alcohol use by the surveyed physicians is also considered a problem that should be addressed by medical educators; only 46 percent felt that moderate or no alcohol use should be encouraged, compared with 93 percent who felt the physician should assume responsibility for helping their patients to quit smoking. See also 83-0687.

84–0284. Viedma, C. **Stop Smoking—With a Smile.** *World Health* p. 29, August 1983.

An antismoking campaign emphasizing the positive aspects of life without cigarettes was recently launched by the Health Studies Institute of the Department of Health and Social Security of Catalonia, Spain. All available audiovisual facilities were used: radio and television spots, advertisements, notices, folders and stickers, and publications oriented toward various socioeconomic levels. Doctors received a handbook on prevention that described psychosocial aspects and medical complications of tobacco addiction, including chapters on passive smoking, health education, legislation, and smoking cessation. Educational brochures and tobacco research results were prepared for teachers, while two publications distributed in schools (a children's story and a comic book) emphasized social consequences (i.e., rotten teeth, bad breath, sensory effects), commercial manipulation, and giving up smoking for positive reasons. The Health Studies Institute is planning to sponsor the First Mediterranean and Southern European Meeting on

Smoking and Health in early December, and a health education campaign for young people in March.

84–0285. Willard, N. **Tobacco: Third World Warning.** *WHO Chronicle* 37(3): 86-90, 1983.

Findings of the WHO Expert Committee on Smoking Control Strategies in Developing Countries suggest that a serious problem exists, since the public is not aware of the dangers of smoking and educational, legislative, or other measures are not being taken to combat the smoking epidemic. Data show that lung cancer is increasing steadily in Hong Kong, and other smoking-related diseases are on the rise in developing countries. In addition, many Third World countries sell cigarettes with high tar and nicotine yields, and produce 63 percent of the world's tobacco (compared with only 50 percent in 1961-1963). While cultivation has become a substantial source of employment, and taxes on tobacco are "politically comfortable" (easy to administer and generally acceptable), they cannot offset the economic losses caused by tobacco production and use: health care expenditures, absenteeism, domestic and forest fires, use of scarce fuel to cure tobacco, and reduced food production (malnourished people are ill-equipped to withstand respiratory and other smoking-related diseases). Governments of developing countries are urged to take the necessary political, social, and educational measures without delay. Action against smoking can be inexpensive, yet effective: (1) health warnings can be placed on cigarette packages; (2) marketing practices can be standardized; (3) taxation can be increased to reduce consumption; (4) alternative land use should be explored in countries growing tobacco; and (5) all tobacco advertising and promotion should be prohibited. Legislation must, however, be linked to public information and education programs, in which the mass media are used to convey the message that not smoking is natural social behavior and should be the social norm.

SMOKING CESSATION METHODS

84–0286. Andre, E. **Animation de Groupe Pour Sevrage Tabagique. [Group Guidance in Smoking Withdrawal.]** In: Molimard, R. (Editor). *1ere Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 95-100, French.

For the last 3 years, group psychotherapy for smoking cessation has been used at the Allevard Health Resort in France. The method was designed primarily for those who have already tried to stop smoking with mixed results, and who want to reduce or to eliminate their smoking habit. Guided groups of 12 to 13 people meet for 2 hours/day for a week. Two working sessions are devoted to discussion and sharing of the emotional experiences of each participant and information about smoking and the bronchopulmonary tract. After the first working session and a nonsmoking day, a discussion is held about smoking dependence and motivation for cessation. Following a day of relaxation, information is provided on cardiovascular pathology and respiratory effects induced by cigarette smoking. During the last session, the problems of weight gain and what to substitute for cigarettes are covered. Those willing to stop smoking are invited to reflect on the relationship between smoking and their daily emotional experiences and the causes of relapse. Emulation within a group and a sense of solidarity are the prerequisites for success.

84–0287. Glasgow, R. E.; Klesges, R. C.; Godding, P. R.; Gegelman, R. **Controlled Smoking, With or Without Carbon Monoxide Feedback, as an Alternative for Chronic Smokers.** *Behavior Therapy* 14: 386-397, 1983.

Results suggest that controlled smoking can be used in a group setting with long-term, heavy smokers; and that carbon monoxide feedback is not essential to treatment success. Sixty chronic cigarette smokers were randomly assigned to one of three treatment conditions: controlled smoking, controlled smoking plus weekly carbon monoxide feedback, or delayed treatment control. The controlled smoking treatments lasted 5 weeks and sequentially focused on achieving 50 percent reductions in nicotine content of brand smoked, number of cigarettes smoked/day, and percentage of each cigarette smoked. Self-monitoring records indicated that subjects in the two controlled smoking conditions successfully reduced each of the target behaviors, and that timing of these changes immediately followed treatment initiation within the multiple baseline across behaviors design. There were few differences between the two treatment conditions, and both treatments produced greater change than the control condition on all targeted behaviors. Carbon monoxide levels were also reduced significantly more in the treatment conditions than in the control condition. Both the carbon monoxide and behavior reductions were maintained at 3- and 6-month followup. (Auth. Abs. Mod.)

84–0288. Lacroix, J. C. **Acupuncture et Dependance Tabagique. [Acupuncture and Dependence on Smoking.]** In: Molimard, R. (Editor). *1ere Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 101-102, French.

The traditional Chinese approach is proposed for researching the process of smoking dependence and the mechanism of acupuncture's effect on smoking withdrawal. A statistical approach was used to develop a therapeutic procedure of smoking cessation by acupuncture, and a previously published followup study of smokers thus detoxified is cited in support. A workplace program administered by qualified physicians and a psychological followup study of ex-smokers is proposed by the Service of Occupational Medicine. In France, 58 physicians who currently practice acupuncture volunteered for service, but the problem of medical ethics is cited as an obstacle. The possibility of detoxifying a smoker in only one or two sessions, at a very low cost, is emphasized.

84–0289. Massonnet, B.; Frison, D.; Llung, L. **Bilan d'une Therapie Comportementale Anti-Tabac. [Results of Anti-Smoking Behavior Therapy.]** In: Molimard, R. (Editor). *1*^{ere} *Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 91-94, French.

A behavioral therapy method for smoking cessation, used with 200 patients in Lyon, France, consisted of creating both progressive aversion to cigarettes and rewards for abstinence. The 1-hour daily outpatient treatment lasted 6 days. During the first half hour, a patient smoked a cigarette and was given unpleasant stimulation in the first session, smoked rapidly under medical control in the following session, and finally was subjected to excess smoke in front of a mirror. The second half of each session was devoted to discussion with the therapist. Followup 1 year later showed that (1) the overall abstinence rate was 33 percent; (2) 43 percent were either abstinent or had significantly reduced consumption; (3) both men and women showed a constant weight gain; (4) 85 percent of the men aged 50 to 60 years relapsed, while women over age 40 showed better results; and (5) relapse was more frequent among blue-collar workers, men with increased responsibility, and depressed women. It is concluded that behavioral therapy has the same success rate as other methods of smoking withdrawal.

84–0290. Pederson, L. L.; Wood, T.; Lefcoe, N. M. **Use of a Self-Help Smoking Cessation Manual as an Adjunct to Advice From a Respiratory Specialist.** *International Journal of the Addictions* 18(6): 777-782, 1983.

A commercial self-help manual was not an effective smoking cessation aid among pulmonary disease patients. All patients were advised to quit smoking by a respiratory specialist. Thirty-five patients were then provided with a manual detailing techniques for quitting; the remaining 40 patients served as controls and were given no additional information or instruction. The cessation rate in the treatment group was 16.7 percent; and in the control group, 25.6 percent (difference not statistically significant). Approximately half of the patients who received the manual used it, but only 12.5 percent of the users found it helpful. It was concluded that providing patients with specific instructions on quitting smoking as presented in the manual did not appear to be useful. (Auth. Abs. Mod.)

84–0291. Toomes, H.; Paul, K. **Raucherentwohnung mit Einem Nikotinhaltigen Kaugummi. [The Effectiveness of Nicotine-Impregnated Chewing Gum in Breaking the Smoking Habit.]** *Praxis und Klinik der Pneumologie* 37(7): 273-277, July 1983, German.

The effectiveness of a nicotine-containing chewing gum (Nicorette 2.0 mg) as an aid in giving up cigarette smoking was tested in 51 unselected persons. Cessation of smoking was motivated mainly by health reasons. After 6 months, one-third of the probands were still nonsmokers and this was confirmed by carboxyhemoglobin determinations. Nicorette had been chewed for an average period of 11 weeks. The failures were mostly attributable to psychological factors, but undesirable effects of the chewing gum, such as sticking to dentures and irritation of the throat, also played a role. Means to improve the success rate are discussed. (Auth. Abs. Mod.)

84–0292. Working Group on Physician Behaviors to Reduce Smoking Among Hypertensive Patients. **The Physician's Guide. How to Help Your Hypertensive Patients Stop Smoking.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, NIH Publication No. 83-1271, April 1983, 27 pp.

A step-by-step approach to smoking cessation counseling, designed for physicians with a busy office practice, is outlined. The minimal procedure can be readily adapted to routine office visits of high blood pressure patients who smoke. Expanded procedures, offering more detailed approaches, are also included for physicians who have the time and interest to devote greater attention to the smoking habits of their hypertensive patients. The four steps of the minimum procedure are (1) asking hypertensive patients about smoking habits; (2) delivering a firm quit smoking message; (3) agreeing on a quit date; and (4) checking on patient's progress at each high blood pressure visit. When minimum procedures are not enough, the physician can offer self-help materials, refer patient to a smoking cessation program, or teach ways to quit smoking. Sample discussions, tips on quitting, sample patient smoking record, and agreement (to quit) form, are included. Information on how to obtain free publications and a kit (posters for office and patient handouts) includes an order form and telephone numbers for the Cancer Information Service.

TOBACCO PRODUCT ADDITIVES

84–0293. Stuetz, D. E. **Smoking Preparations.** United States Patient No. 4,397,321, August 9, 1983, 6 pp.

Tobacco and nontobacco smoking compositions that deliver smoke of reduced tar and nicotine content, and with a substantially reduced carbon monoxide content, are described. A catalyst composition, consisting of fine ash and a transition metal compound, is dispersed throughout the filler. Up to 10 parts by weight of both the ash and transition metal components are recommended. Under controlled test conditions, a 37 percent conversion of carbon monoxide to carbon dioxide in the mainstream smoke was achieved. (Auth. Abs. Mod.)

TOBACCO MANUFACTURING AND PROCESSING

84–0294. Baker, R. R.; Dashley, F. J. **Smoking Articles.** United States Patent No. 4,407,308, October 4, 1983, 4 pp.

The described smoking article is comprised of a rod of smoking material with a wrapper that has a substantially uniform air permeability due to viscous flow of not more than three and preferably not more than two Coresta Units. Furthermore, the ratio of the coefficient of diffusion of oxygen through nitrogen in the paper and the thickness of the paper (Do/t value) should be between 0.08 to 0.65 cm/sec, preferably 0.15 to 0.25 cm/sec. The length of the rod of smoking material may suitably be within a range of 25 to 55 mm. A reduction in sidestream total particulate matter delivery of 40 percent or more can be obtained. The invention is also concerned with a smoking article wrapper material which satisfies the above permeability and Do/t value requirements. (Auth. Abs. Mod.)

84–0295. Haynes, R. L. **Carbon Monoxide Poisoning From Non-Tobacco Cigarettes.** *Journal of the Medical Association of Georgia* 72(8): 553-555, August 1983.

Substitution of nontobacco cigarettes may be associated with more immediate health hazards (i.e., carbon monoxide poisoning) than tobacco smoking, despite the known dangers of tobacco cigarettes. Five hospital volunteers (three females and two males, aged 22 to 42 years, all regular smokers of one to two packs tobacco cigarettes/day) smoked 20 of their usual cigarettes over 12 hours or less and, on the following day, smoked 20 nontobacco, cocoa bean-derived cigarettes (Free) over the same time period. Venous carboxyhemoglobin (COHb) levels were measured at 15, 60, and 180 minutes following completion of the last cigarette on both days of testing. For each measurement period, the nontobacco product achieved significantly higher COHb values than tobacco; no participants showed lower COHb values with the tobacco substitute than with tobacco. The two males achieved COHb values of 17.6 and 16.5 percent, measured 15 minutes after cessation of smoking. Smokers with higher tobacco-associated COHb values demonstrated that their habits of inhaling more smoke could produce striking elevations with the nontobacco product. Subjects showed mean COHb values of 8.26 ± 1.08 percent 15 minutes after smoking the last of 20 tobacco cigarettes, compared with 13.08 ± 1.69 percent 15 minutes after the last nontobacco cigarette. Values remained significantly above the corresponding tobacco values at the 2- and 4-hour sampling periods. While signs of carbon monoxide poisoning are described, it is noted that unconsciousness may occur without additional symptoms. It is stated that exposed nonsmokers will develop COHb elevations from sidestream smoke.

84–0296. Hoffmann, D.; Adams, J. D.; Haley, N. J. **Reported Cigarette Smoke Values: A Closer Look.** *American Journal of Public Health* 73(9): 1050-1053, September 1983.

The effects of mechanical compression of filters and blocking of air channels on smoke yield in seven brands of filter cigarettes were investigated. In addition, the influence of these variables on actual uptake of smoke constituents by smokers was studied with four subjects. Filter compression produced major increases in smoke yields in a cigarette featuring a filter tip with four longitudinal air channels at its periphery. Blocking the air channels increased tar yield by 51 percent, nicotine by 69 percent, and carbon monoxide by 147 percent. Subjects who smoked this cigarette tended to smoke fewer cigarettes/day than when smoking cigarettes with perforated filter tips, yet plasma cotinine levels were significantly higher. Blood pressure and pulse rate were markedly elevated after first exposure to smoke from the special filter cigarette, as were plasma nicotine levels. Thus, individuals inhale different quantities of smoke constituents from cigarettes with reportedly similar smoke yields as defined by Federal Trade Commission methods. Redefinition of "average" smoking parameters and adjustment of standard laboratory methods are suggested. (Auth. Abs. Mod.)

84–0297. Institute of Paper Chemistry. **Cigarette Filters: Composition and Manufacture (1972 - Mar 83). Citations From the Institute of Paper Chemistry Data Base.** U.S. Department of Commerce, National Technical Information Service Publication No. PB83-862045, March 1983, 123 pp.

A bibliography containing 109 citations concerning processes and apparatus used in the manufacture of tobacco smoke filters for cigars and cigarettes is provided. Synthetic and natural fibrous nonwoven web materials are evaluated for cigarette smoke filtering capabilities. Performance of filtering webs is also examined with reference to reinforcing impregnations and aromatic additives. Developments in equipment and manufacturing techniques for processing cigarette filters are included. Ordering information for individual reports, a sample citation for user reference, and a title listing by page number precede the abstract section. A majority of the citations are descriptions of foreign and domestic patents. A subject term index with page and citation reference number completes the report.

84–0298. Lamb, C. G. **Cigarette Filter.** United States Patent No. 4,406,294, September 27, 1983, 6 pp.

The described cigarette filter includes a porous filter rod circumscribed by an air-permeable wrapper, and embedded with longitudinal ventilating grooves. The wall of the grooves is impermeable to air and smoke, while a portion of the remaining area of the wrapper is air-permeable. Porous tipping material circumscribes the filter rod, providing a means for air to enter both the filter rod and the grooves. The air flowing through the filter rod dilutes the smoke before it reaches the smoker's mouth, while the grooves deliver only air. Thus both ventilation and dilution are used to lower tar while achieving relatively normal pressure drop.

84–0299. Llanos, M. **Improvements in Reducing Risks to Smokers.** *Tabak Journal International* 4: 266, 268-269, August 1983.

In 1956, the average tar content of U.S. cigarettes was 38.4 mg. By 1964, average levels had dropped to 23.1 mg, and reached 13.2 mg tar in 1980. The Federal Trade Commission reported that of 176 brands on sale in 1979, 103 yielded less than 15 mg tar, and 101 delivered less than 1 mg nicotine. Similar trends are reported in the United Kingdom. Methods that effectively improve the quality of cigarette smoke from a biological perspective are discussed, including leaf cultivation, processing, and curing; filters and filter additives; and tobacco additives (including some that lower combustion temperature). It is noted that the appropriation of these methods will depend on the economic results. In general, demand for mild, low-tar products is highest among women, whites, and those in upper educational and income brackets. Youth appear to be less inclined toward low-yield cigarettes.

84–0300. Tobacco Reporter. **Refined Technology for Controlled Delivery and Higher Speeds.** *Tobacco Reporter* 110(8): 40-41, August 1983.

Although there was a 7 to 10 percent decline in total cigarette consumption in Great Britain over the last year, the preference for light (low-tar, filtered) cigarettes is increasing. Changes in cigarette-making speeds, from 4,000 to 6,000/minute presently to 10,000 in the next generation of technology, will require papers, adhesives, plug wraps, and other components that will accommodate both industry standards and the need for precise control of cigarette smoke delivery. The trend toward invisibly pre-perforated tipping continues; an estimated 25 percent of cigarettes sold in Britain will have tip ventilation by the end of the year. However, consumer trends show a return to more flavorful, higher tar products, and the industry will rely increasingly on selective filtration rather than dilution in this market segment. New instrumentation is also reported, including Filtrona's PPM 300 and RRM 500, which monitor paper uniformity and permeability; the PZM 300 that surveys plasticizer application rates; and the AVP and AVM series that measure pressure drop.

84–0301. Townsend, D. R. **Cigarette Ventilation: Focus on the Filter.** *Tobacco Reporter* 110(8): 42-43, August 1983.

Based on the expectation that demand for low-yield cigarettes will increase, various types of filters that will dilute the smoke while retaining taste and adequate pressure drop to the smoker are described. Ventilated double filters of acetate or acetate/cellulose, and the CPF filter, which has an inner core wrapped in a grooved or fluted porous material along part of its length, are already in use. Newer types, such as the ASTRA, are based on the assumption that enhanced taste is achieved by not allowing the smoke to mingle with the ventilation air before they both reach the smoker's mouth. In the ASTRA, the inner dual acetate core is wrapped in an inner nonporous wrap and again in a special grooved impermeable material with inner flutes. When surrounded by tipping, outer flutes are created, along which air passes from perforations in the tipping. A high proportion of the tobacco smoke will be confined within the filtration core and will not mix with ventilating air until both enter the smoker's mouth. Two other new filters, which are designed to reduce carbon monoxide in relation to tar at acceptable pressure drop levels, are also described.

TOBACCO ECONOMICS

84–0302. Bergamin-Veragut, L. **Decline in Production of Swiss Tobacco Goods.** *Tabak Journal International* 4: 296, August 1983.

Production of tobacco goods in Switzerland declined slightly from 1981 to 1982. As in previous years, the products most often affected were "Stumpen" (cheroots) and pipe tobacco. Cigarette production maintained its usual level, although a few individual brands sustained losses. The tourist trade, exports, and frontier traffic helped to sustain production. Production and consumption data are provided for cigarettes (Maryland, American Blend, European Blend), cigars (cheroots, cigarillos, "Kopfzigarren," Toscani, Virginia), pipe tobacco, plug tobacco, and snuff. (Auth. Abs. Mod.)

84–0303. Burry, R. D. **Declining Cigarette Shipments May Prompt Uncertain New Products Moves.** *Tobacco International* 185(15): 92, 94, 98, July 22, 1983.

More important to cigarette manufacturers than the actual 4.9 percent decline in consumption for the first quarter of 1983 is the determination of which portion of the decline is temporary (shock over sharply higher retail prices) and which is permanent. Early indications are that the domestic industry probably will not return to a rising demand trend. The industry's pricing environment is equally uncertain; manufacturers began uncoordinated increases in August 1982 to offset anticipated Federal excise tax increases, and a recent increase of 1.5 dollars/1,000 units is the largest ever imposed and exceeded expectations. Producer prices (Philip Morris) for 1,000 king-size units show a year-to-year percent change of 9.3 percent in 1980 compared with a 34.0 percent change for the first 6 months of 1983. New product introductions indicate the industry is moving toward European-type segmentation based on a wide spectrum of image/price relationships. There is major domestic uncertainty, however, created by the necessity of basing segmentation almost entirely on image rather than "real" product characteristics.

84–0304. Hill, D. **High Cost of Cigarettes in U.K. Encourages Switch to Roll-Your-Own.** *Tobacco International* 185(14): 13, 16, July 8, 1983.

The roll-your-own market showed slightly declining trends in the United Kingdom until 1981, when a steep tax increase and a continuing recession combined to boost handrolling tobacco sales by 11 percent. Data for 1982 show that handrolling tobacco accounted for approximately 6.2 kg manufactured weight, representing 6.5 percent of total domestic weight. Manufacturers have used price cuts and special offers to encourage sales. Factors contributing to the appeal of handrolling include higher cigarette costs (current price/pack for the most popular brand is 2 dollars and 50 cents), improved packaging with greater shelf life and visual appeal, sustained promotional activity, and a widening range of brands. A brief history of the roll-your-own movement is provided.

84–0305. Hill, D. **Price-Conscious, Export Minded U.K. May Switch From Traditional Sources.** *Tobacco International* 185(15): 58, 60, July 22, 1983.

Reduction in domestic sales has forced most cigarette manufacturers to expand exports, which reached 46,392 million pieces in 1982, compared with only 28,730 million units in 1977. Carreras Rothmans exported two-thirds of its total sales to 165 countries, while the percentage for tobacco products of total consumer expenditures domestically fell from 4.2 percent in 1977 to 3.6 percent in 1982, with the current figure probably even lower. Total domestic sales (102 billion units) were the lowest since 1962 and represent the eighth successive annual drop in consumption levels. In addition, in accordance with voluntary agreements with the government, manufacturers have spent large sums to develop and promote low-tar brands, which now account for about 53 percent of sales and two-thirds of advertising costs. The aim of reducing the average tar yield of all cigarettes sold in Great Britain to 15 mg by the end of 1983 has probably already been met. The market is committed to flue-cured (90 percent of imported unmanufactured tobacco) and manufacturers have relied on U.S. tobacco (the costliest major source). However, new sources of good quality, reasonably priced leaf are being sought, and Brazil is now the major supplier.

84–0306. Hill, D. **High Exports Level Cushions Slumping Domestic Market in the Netherlands.** *Tobacco International* 185(15): 61, 64, July 22, 1983.

The fortunes of the tobacco companies in the Netherlands reflect the difficulties being experienced throughout the economy. Slow growth of consumer incomes and higher product prices have made it difficult to maintain sales, particularly when faced with a sustained antismoking campaign. While the domestic market is slightly better than in 1981, exports of cigarettes in 1982 were more than double import figures, demonstrating the popularity of Dutch tobaccos. Cigar and manufactured tobacco exports were also higher than imports (6,938 tons of manufactured tobacco imported compared with the export of 28,888 tons). For Dutch manufacturers, the key to success is to continue the historically high level of exports, especially cigars, which have a premium value in many markets.

84–0307. Lalande, L. **France Introduces a Supplementary Tax on Tobacco Goods by Instalments.** *Tabak Journal International* 4: 290-291, August 1983.

A 25 percent supplementary tax on tobacco products, due to take effect in April 1983, was postponed due to massive protests from tobacconists, growers, and industry representatives. It was pointed out that 10,000 people were employed in processing tobacco, 26,000 in growing tobacco, and 45,000 retailers in selling the products. If the tax resulted in only a 15 percent decline in consumption, there could be a loss of 500 jobs, and the smaller farms would suffer the most. In March, it was announced that the tax would be introduced in installments (5 percent every 6 months) beginning in July 1983. The tobacco trade will probably be able to cope with the modified tax, but it is still not in accordance with regulations specified by the European Community. The French Government must thus expect countermeasures to be taken. A 25 percent tax on alcoholic beverages, introduced with the tobacco tax, took effect as planned in April 1983, although opponents noted that there was no evidence that price increases reduced consumption among addicts.

84–0308. Maxwell, J. C., Jr. **Maxwell Report: Smokeless Grows; Cigars Decline.** *Tobacco Reporter* 110(8): 56-57, August 1983.

While the total volume of chewing tobacco declined 0.3 percent in 1982 (with the largest decreases in the loose leaf/moist plug categories oriented toward older blue-collar workers), the moist snuff/fine cut categories gained more than 8 percent. It is stated that the younger generation may be initiating consumption in this area rather than cigarettes, and demographics are skewed toward the younger age groups. While the decline in the cigar industry accelerated in 1982 (perhaps due to the economy), less decline and upward trends are anticipated. Volume of high-price (50 cents or more) cigars rose 5.2 percent in 1982 to 13.9 million units. Smokeless tobacco market statistics for 1981 and 1982 are given for (1) production and percent of market share by company and brand; (2) total smokeless volume and market share by company; and (3) market share for both large and little cigars by units and percent for companies and brands.

84–0309. Maxwell, J. C., Jr. **Smokeless Keeps Growing; Cigars Keep Declining.** *Tobacco International* 185(15): 90-91, July 22, 1983.

Chewing tobacco overall volume declined 0.3 percent in 1982, with the greatest decrease occurring in the plug categories. However, moist snuff/fine-cut products gained more than 8 percent. The younger generation may be consuming moist snuff rather than cigarettes as the demographic makeup is skewed toward younger groupings. The plug sector of the market is tied to older blue-collar workers and is thus related to jobs and the economy, suggesting that plug sales may pick up as the economy improves. The decline in cigar sales accelerated in 1982, except for the high-price cigars (≥50 cents), which jumped 5.2 percent to 13.9 million units. The market shares of little cigars, smokeless tobacco products, and large cigars are tabulated by brand for 1981 and 1982.

84–0310. Maxwell, J. C., Jr. **How the Brands Ranked. Maxwell International Estimates.** *World Tobacco* 82: 50-52, September 1983.

Tabulated cigarette data for 12 countries (United States, West Germany, Great Britain, India, Pakistan, Poland, Australia, Greece, Netherlands, Chile, Costa Rica, and Cape Verde Islands) cover market division by type (filter, nonfilter) and market share for 1980, 1981, and 1982 (in most cases). Also listed are positions of leading brands by brand/type and manufacturer, with market shares for corresponding years. Total sales (million pieces) are provided. This compilation, when combined with figures published in the June 1983 issue, covers most of the world's cigarette trade.

84–0311. Misdorp, S. **Argentine Virginia, Burley Crops Larger; International Brands Gain.** *Tobacco International* 185(13): 33-34, June 24, 1983.

In Argentina, the tax on cigarettes was increased from 70 to 75 percent in May 1982, but was removed in January 1983 and sales increased 4.2 percent between January and May 1983. However, the chronic economic situation has forced manufacturers to introduce brands containing only 10 cigarettes/pack, which now make up 10 percent of the market. Sales of dark tobacco and American blend cigarettes have leveled off. Long-term trends are favorable for international brands, although domestic brands remain popular for economic reasons. Low-tar sales are stable and roll-your-own sales are down. The emphasis in leaf production is toward higher quality (nicotine levels averaged 2.5 percent in Jujoy and 2.0 percent in Salta for Virginia, and 3.0 percent in Santa Fe and 1.3 percent in Salta for Burley). Prices, crop problems, and planting trends are also reported.

84–0312. Peter, R. **Uruguay's Tobacco Industry Demands Measures Against Smuggling.** *Tabak Journal International* 4: 256-257, August 1983.

Owing to the high tax rate on tobacco, Uruguayan tobacco products are between 70 and 150 percent more expensive than those in neighboring countries. In spite of strict control measures at the borders, this has led to a constant increase in illegal imports and it is estimated that about 30 percent of domestic consumption is already being met by smuggled goods. To reduce smuggling more effectively, the Uruguayan manufacturers and importers of tobacco goods recommend a considerable decrease in the tobacco tax and emphasize that this decrease should be larger in the border provinces than elsewhere. Furthermore, attention is drawn to the serious losses being sustained by the government in tax revenue and to the jobs that are at risk. (Auth. Abs.)

84–0313. Seuss-Calandrelli, D. **Move Towards Light Cigarettes in Italy.** *Tabak Journal International* 4: 292, 294, August 1983.

Tobacco consumption in Italy rose 5 percent over the last 3 years, mostly in domestic products, due to the pricing policies of the tobacco monopoly. Prices of Italian tobacco products are the lowest in the European Community. Total sales were 103,136,000 kilos in 1982 compared with 102,984,000 kilos in 1981, providing a 32 percent increase in revenue that was 25 percent of state income from national taxation. Changes in consumption patterns, due particularly to the influence of women, toward European-blend or light cigarettes, are reflected in production and sales of new Italian products. The Futura brand should win a fair share of the new market, 60 percent of which is currently held by manufacturers of foreign products.

84–0314. Stone, M. **Overseas Memo. Evidence Supports Advertising.** *Tobacco Reporter* 110(9): 6, September 1983.

Evidence is presented to show that banning the advertising of tobacco products does not reduce the incidence of smoking. In three East European countries (Hungary, Poland, and Bulgaria), which have a total ban on advertising, consumption increased at three to five times the rate found in the United States and the United Kingdom. It is suggested that such bans may be the wrong approach for curtailing consumption, and may well limit any positive benefits that might accrue. The proportion of smokers using filtered cigarettes in countries banning advertising is about 65 percent, while in other countries where advertising is allowed, the proportion is as high as 90 percent. It is concluded that there is no evidence to suggest that advertising causes people to smoke, and that every country that has tried to reduce smoking by restriction of advertising has been unsuccessful.

84–0315. Tabak Journal International. **Tobacco Industry in Focus.** *Tabak Journal International* 4: 312, 314, August 1983.

The Bureau of Industrial Economics reports that the value of shipments of cigarettes, cigars, smoking tobacco, and smokeless tobacco products increased an estimated 3 percent from 1981 levels. Stemming and redrying shipments were down about 2 percent, amd a decline of the real value of shipments of all manufactured tobacco products is expected to continue in 1983. Cigarettes represent about 92 percent of the value of all 1982 shipments. Cigarette consumption in 1982, which was static due to unemployment and reduced consumer income, totaled 31.6 thousand million packs; however, low-tar cigarettes seem to be topping out. Overall cigarette consumption among smokers aged 18 and older is down slightly, and is not expected to reach 1981 figures. U.S. cigar consumption continues to decline (by about 5 percent in 1982), while value of cigar industry shipments declined more than 3 percent. Consumption of smoking tobacco for the financial year ending June 30, 1982, was 34 million pounds (representing an all-time low), but smokeless tobacco product consumption increased modestly. About two-thirds of the 59,000 manufacturing workers (unchanged from 1981) are employed by cigarette makers. Producers are spending increasing sums on advertising and promotion (600 million dollars for cigarette advertising in 1982), with greater use of couponing, point-of-sale music, and sponsorship of spectator sports events to promote cigars and smokeless tobacco products. The first major change in tobacco marketing regulatory programs since the 1960s, enacted in 1982, requires that price support and production adjustment programs operate at no net cost to the taxpayer (except for administrative expenses). At 25 cents/package, Wisconsin has the highest State tax rate. Tobacco industry-related legislative actions, trade, and statistics are summarized.

84–0316. Tabak Journal International. **Cigarette Industry Faired Well in 1982.** *Tabak Journal International* 4: 298-300, August 1983.

For the U.S. cigarette industry, 1982 was a year marked by limited brand activity, cost-saving measures, state and Federal tax increases, an unsettled economy, and a slight, overall consumption decline. According to the Tobacco Merchants Association, the six major cigarette companies introduced five new brand names and nine line extensions (including packaging variations). Despite these conditions, many companies increased sales, and some increased market shares and initiated extensive modernization or expansion programs. Philip Morris led the industry, as it has for the past 16 years (unit sales up 2.5 percent over 1981 and an increase from 31.8 percent to 32.8 percent of U.S. market share). R. J. Reynolds had the highest unit volume in 1982 (208.9 thousand million cigarettes sold) and its market share grew from 33.1 percent in 1981 to 33.5 percent in 1982 (the largest market share of any U.S. tobacco company since 1963). Despite a drop in unit sales and a 0.6 percent decline in total market share, Brown and Williamson's sales revenues rose 7.7 percent for the year; the company retained its position as third largest cigarette manufacturer in the United States. Sales data for the domestic and international markets of these companies (and their brands), as well as American Tobacco, Lorillard, and Liggett and Meyers are provided.

84–0317. Tabak Journal International. **Interest in Smokeless Tobacco Continues to Increase in Sweden.** *Tabak Journal International* 4: 282-283, August 1983.

The Swedish Tobacco Company annual report for 1982 indicates increased sales of cigarettes, smoking tobacco, and smokeless tobacco, while the cigar market (dominated by cigarillos) declined 3 percent. Cigarette sales increased 5 percent to 12.1 thousand million pieces, largely due to inventory purchases before year-end in anticipation of a scheduled tobacco tax increase. Domestic cigarette sales are expected to decline in 1983. A 17 percent increase in smoking tobacco sales is attributed to intensive border trade with Norwegian consumers, while interest in smokeless tobacco continues to increase. Chewing tobacco sales rose 4 percent (to 23 tons), and sales of smokeless tobaccos were also up 4 percent (to 3,930 tons). Tabulated data for production, sales, imports, and forecasts for cigarettes, cigarillos and cigars, smokeless tobacco, and smoking tobacco are provided.

84–0318. Tabak Journal International. **Canadian Tobacco Industry Is Up to the Challenge.** *Tabak Journal International* 4: 280-281, August 1983.

The fiercely competitive Canadian tobacco industry has met the challenges posed by last August's killing frost (that damaged a third of Ontario's projected 107 million kilo flue-cured crop), a sluggish economy, an increasing tax burden, and a phenomenon of many price-conscious smokers switching from factory-made cigarettes to roll-your-own. This market shift increased sales of domestically produced fine-cut tobacco by 16.6 percent in 1982, while shipments of factory-made cigarettes decreased by one-third of 1 percent. The Canadian tobacco industry proved to be resilient, with estimated sales of 3.9 thousand million dollars in 1982, an increase of 11 percent over 1981. (Auth. Abs. Mod.)

84–0319. Tabak Journal International. **Statistics.** *Tabak Journal International* 4: 331-332, 334, 336-337, August 1983.

Cigarette exports and production, and the percentage of filtered cigarettes compared with total production, are presented for selected countries worldwide. Exports are totaled for 1973 through 1977 and listed yearly from 1978 through 1982. In million pieces, Bulgarian exports showed steady growth with 1982 figures totaling 75,300, slightly higher than United States exports, which declined from a 1981 high of 82,582 (after steady growth) to 73,585 in 1982. United Kingdom exports, showing steadily growing trends, ranked third worldwide with 46,392 million pieces, followed by West Germany and the Netherlands with 38,192 and 35,563, respectively. Tabulated cigarette production data show a world total of 4,578,406 million pieces, just 1,690 above the 1981 total. Mainland China led 1982 world production with 900,000, followed by the United States with 694,200, the Union of Soviet Socialist Republics with 375,000, and Japan with 308,700. World production totals show steady growth, while a comparison between continents showed Asia with the highest production (1,826,860 million pieces).

84–0320. Tabak Journal International. **Aromatic Tobaccos Gaining Favour Among Britain's Pipe Smokers.** *Tabak Journal International* 4: 271, 274, August 1983.

Sales of pipe tobacco in Great Britain have fallen from 5 million kg in 1975 and 1976 to only 3.5 million kg in 1982, attributed to constantly rising cost due mainly to increases in tobacco taxes. However, aromatic pipe tobacco has become increasingly popular, and is expected to hold 25 percent of the market within a few years, up from 14 percent at present. Several reasons may explain this trend: (1) attractive packaging that appeals to younger smokers; (2) freshness for longer periods; and (3) relative mildness. Mildness is particularly important for cigarette smokers who switch because of the higher cost of cigarettes. Advertising on television has also helped promote the image and attract first-time pipe smokers. While imported brands now make up a large part of the market and are subject to fluctuations in currency exchanges, new domestic brands being introduced are expected to stabilize the pipe tobacco market.

84–0321. Tobacco Institute. **The Tax Burden on Tobacco. Historical Compilation.** Washington, D.C., The Tobacco Institute, Volume 17, 1982, 196 pp.

The history of tobacco taxation in the United States, traced from 1865 to 1982, through four pages of charts and 184 pages of tables, is in two sections: (1) composite figures on Federal and State excise taxes on cigarettes and other tobacco products; and (2) history and current cigarette tax status by State. Federal, State, municipal, and total tax year highlights are presented for 1982. Overall data show that excise taxes on cigarettes for the fiscal year ending June 30, 1982, totaled over 6.7 billion dollars, or over 29 dollars for each man, woman, and child. Federal excise taxes for the same period (8 cents/pack, increased to 16 cents/pack January 1, 1983) grossed almost 2.5 billion dollars, while State taxes (ranging from 2 to 25 cents/pack) yielded over 4 billion dollars. Local taxes (ranging from 1 to 15 cents/pack, imposed in 369 towns and cities) grossed 166.4 million dollars. Combined Federal and State excise taxes accounted for approximately 26.8 percent of the retail price of cigarettes; totals for taxes imposed at the three levels of government comprised about one-third the price of each pack sold. Also discussed are taxation effects on growers, manufacturers, and allied industries; sales; and bootlegging.

84–0322. Tobacco International. **Burley May Increase in Paraguay If Drying Machinery Is Imported.** *Tobacco International* 185(13): 42, 44, June 24, 1983.

While planted area for the 1983 Paraguay crop is estimated at 14,000 ha, tobacco production increased to only about 14,000 metric tons (farm sales weight) due to cold weather and excessive rain, with reduced quality expected. Since drying facilities for Burley tobacco will not be available before 1984, dark tobacco continues to be the main type produced although light cigarettes are gaining in popularity. Domestic industrial use of tobacco is estimated at 1,600 metric tons for 1982, with 80 to 90 percent allocated for cigarette manufacturing. Production has declined from 37.8 million packs in 1981 to 35 million packs (about 50 percent filters) estimated for 1982, probably attributable to the economic recession. Also provided are data for imports, cigarette production, unmanufactured tobacco exports (1981 to 1982), and unmanufactured tobacco supply and demand (1981 to 1983).

84–0323. Tobacco International. **Blue Mold Harder on Cigar Than on Cigarette Leaf in Honduras.** *Tobacco International* 185(13): 38, 41, June 24, 1983.

This is the best year for cigar tobacco producers since blue mold arrived in Honduras in 1979. Estimated tobacco production for 1982 to 1983 is 4,199 metric tons (dry weight equivalent), a 4 percent increase over 1981-1982 production of 4,048 metric tons. High Burley production is due to increased area, while higher cigar tobacco production is the result of better yields. Production of cigarette tobacco is expected to be about the same as it was last year, due to a low world market price for Burley and the stagnant local cigarette market. Cigarette consumption showed a steady rise from over 107 million packs in 1980 to more than 114 million in 1982. However, a generally unfavorable economic climate and a price increase from 37.5 cents/pack in 1982 to 40 cents/pack in 1983 (for the brand that accounts for 50 percent of consumption) may cause sales to decline.

84–0324. Tobacco International. **Austrian Monopoly's Production Up; Foreign Sales Increase in 1982.** *Tobacco International* 185(15): 50, 52, 56, July 22, 1983.

Production of tobacco in Austria (controlled by a monopoly) declined 3.3 percent in 1982 to 421 metric tons (MT) while imports of raw tobacco totaled 11,012 MT, 20 percent less than 1981 (the amount of U.S. tobacco imported increased by 0.7 percent). Consumers continue to prefer cigarettes that contain a significant proportion of U.S. tobacco, which cost 6,830 dollars/MT in 1982 compared with 2,789 dollars/MT for non-U.S. leaf. The monopoly encourages the cultivation of tobacco in marginal farming areas, and as an alternative for crops that have been overproduced without adequate available outlets. Although cigarette production increased nearly 6 percent, domestic sales declined except for filter-tipped (0.4 percent gain), which now constitute 96.4 percent of the market. Cigarette exports increased slightly, particularly to Poland, Saudi Arabia, Indonesia, Greece, and Yugoslavia. Imports declined by 15.2 percent (17.4 percent for U.S. cigarettes). Investments continue to be made in new buildings and machinery, but the monopoly also intends to diversify into other industries.

84–0325. Tobacco International. **Cigarette Report 1982 1983.** *Tobacco International* 185(15): 38, 40, July 22, 1983.

The Italian tobacco monopoly revenues were 32 percent higher in 1982 compared with 1981 (98 percent from sales of cigarettes, 2 percent for all other products). Since 1980, the trend toward foreign brands has reversed, with Italian brands selling 5.5 percent more in 1982 than in 1981, mostly due to price differences. Holland (Philip Morris brands) is the largest supplier of imported cigarettes, and, with Germany, accounts for 95 percent of all imports. Revenues for domestic brands were up by 43 percent over 1981, compared with only a 20 percent increase in revenues from imports. In July 1983, prices of all cigarettes were increased substantially; the most popular Italian brand now costs 1,300 lire compared with 2,000 lire for imported Marlboro, the second best-selling brand. Revenues for the first 6 months of 1983 were up by 17 percent, but estimates of cost increase effect on consumption are not provided. Overall quantity for the period was 49,049 metric tons, a drop of 1 percent.

84–0326. Tobacco International. **In France: Blond Sales, Growth and Processing of Light Tobaccos Gain.** *Tobacco International* 185(15): 42, 44, 46, 48, July 22, 1983.

Total cigarette consumption in France increased 1.1 percent in 1982, counter to a slight downward trend in recent years, partly due to improved sales of "lighter" cigarettes. The trend toward light tobacco production continued in 1982, although dark tobacco still represented more than 92 percent of area planted. Because of the increasing demand for American-type (light tobacco) cigarettes, the government is providing financial incentives for farmers who switch to the light tobaccos. Total production of cigarettes (47 percent filtered) remained stable while production and sales of other products continued to decline; cigar sales were down 3.2 percent in 1981 and 7.4 percent in 1982. The government's share now represents 74.5 percent of the retail price of cigarettes following a 5.2 percent price increase last year. An additional 26 percent increase in July 1983 is expected to lead to an 8 to 10 percent decline in domestic consumption that could be higher for the light cigarettes, which are more expensive. Tobacco growers are constructing a processing plant for blond tobacco (the first in France) with government aid, and agreements with an Italian trading company are expected to improve foreign markets. Imported American-type cigarettes are expected to be 50 percent of sales by 1985, with about a third of the light tobacco being supplied by the United States.

84–0327. Tobacco International. **Sales of Generic Cigarettes Grow 200 Percent; Hold 2.5 Percent of Market.** *Tobacco International* 185(15): 112, July 22, 1983.

The doubling of the excise tax on cigarettes in January, and an aggressive marketing campaign by the manufacturer of 96 percent of the generic brands sold in the United States, are responsible for the 200 percent increase in generic sales in 1982. The market share for generics, now at 2.5 percent, is expected to go higher as industry price structures make discounts more meaningful; generics sell for about 2 dollars less/carton. In addition, reverse snob appeal may be a factor with consumers refusing to be swayed by advertising and demonstrating price and quality consciousness. Market research has shown that nearly 85 percent of those who try generics like them as well or better than their current brand and 94 percent would buy them again. Six kinds of cigarettes are available: Lights 85 and 100 mm, menthol and nonmenthol; and Ultra Lights 85 and 100 mm, nonmenthol.

84–0328. Tobacco International. **Portugal to Modernize Its Cigarette Manufacturing, Expand Its Exports.** *Tobacco International* 185(15): 78, 81, July 22, 1983.

Because of antismoking activities and a 20 percent increase in cigarette prices in Portugal, combined sales of cigarettes rose only 4.2 percent in 1982 (4.6 percent for filtered cigarettes and 2.2 percent for nonfiltered). Sales by the state-run monopoly provided 6.4 percent of the government's revenue for 1982. Areas under cultivation for flue-cured were up 26 percent, and there was a threefold increase in the output of Burley tobacco. Manufacturers' requirements (up 6 percent) are expected to decline as even higher prices for cigarettes will undoubtedly have an adverse effect on consumption. Since only a fixed amount is allocated yearly for imports, the prospects for U.S. market development activities are not good (Portugal's policy is to buy from the cheapest source, creating an erratic buying pattern). Large investments have been made to modernize curing facilities and manufacturing plants. New brands are being created for export to international markets (mainly Europe) in cooperation with state-run industries in other European countries and Japan. Exports now are mainly to Portugal's former African territories.

84–0329. Tobacco International. **In Spain, Mechanization Still Lags, American-Blend Cigarettes Gain.** *Tobacco International* 185(15): 67, 71, 74, 76, July 22, 1983.

While the marketing of tobacco in Spain is highly organized, controlled by the Tobacco Cultivation and Fermentation Service through permits, mechanization is limited (although encouraged) and most tobacco is still picked by hand. Acreage is expected to increase, but inadequate irrigation water may reduce yields. However, price increases in 1983-1984 are expected to be moderate, and the government will probably continue to provide free seed (except flue-cured) and other incentives. A decline of about 1 percent in tobacco consumption is expected in 1983 due to an increase in the price of cigarettes, static economic conditions, and a decrease in the tobacco used in each cigarette. Use of leaf from the United States is expected to increase, as smokers continue to switch from dark to American-type cigarettes. Spain is also expected to export 55 percent more unmanufactured tobacco as quality improves and the price remains relatively low. Filtered cigarettes account for about 90 percent of output, but low-tar/ low-nicotine cigarettes comprise only about 2 percent. Tabulations are provided for 1982 imports and exports of unmanufactured tobacco, cigarettes, and cigars.

84–0330. Tobacco Reporter. **Spotlight on Eastern Europe: Money and Materials Remain Critical.** *Tobacco Reporter* 110(8): 44-47, August 1983.

Tobacco production status is reported for Yugoslavia, Poland, East Germany, and the Soviet Union. Cigarette production declined by an estimated 3.2 billion pieces in Yugoslavia (to 61 billion), with filter cigarettes accounting for 97 percent of total production. Shortages of supplies remain a problem in Poland. While cigarette rationing was discontinued in March 1983, prices increased and the scarcity of high quality cigarettes, combined with the tight money situation, will lead to a decline in sales (around 90 billion pieces in 1983 compared with 92 billion in 1982). Emphasis in East Germany is on self-sufficiency (85 percent of tobacco is imported) and a reduction in consumption. However, cigarette sales continue to climb; roughly one-third of adults and one-half to three-fourths of youth smoke. In 1982, sales reached 295 billion pieces, with average consumption at 15 cigarettes/day. In the Soviet Union, 1983 cigarette production is planned at 373 billion pieces, but leaf output remains below the production quota, implying a reliance on imports to meet requirements.

84–0331. Tobacco Reporter. **Generics Update.** *Tobacco Reporter* 110(8): 64, August 1983.

In the United States, sales of generic cigarettes, which increased by 200 percent during 1982, claimed 2.5 percent of the total market, picking up much of the slack caused by the doubled Federal excise tax. In contrast, generic cigarette sales in West Germany lost ground rapidly as major manufacturers introduced cheaper brands and made extensive price adjustments following the July 1982 tax increase. Generic sales reached 807 million pieces in December 1982 (8.6 percent market share in January) but then dropped to 510 million (5.4 percent of market) by May 1983. However, the combined market share of the cheaper brands and generics rose from 9.24 percent in January to 36.07 percent in May (total 3.4 billion pieces) at the expense of the traditional brands, which fell 20 to 40 percent overall. German cigarette makers plan to increase the price of their cheap brands and lower the cost of the expensive ones to attract smokers back to the established brands.

84–0332. Tobacco Reporter. **Western Europe: Race to Regain Shares.** *Tobacco Reporter* 110(9): 44, 46, September 1983.

In response to last year's 30 percent West German cigarette tax increase and cigarette market share losses to both generics and fine-cut tobacco, German manufacturers have reduced prices of traditional best-selling brands by 5 percent, reduced the number of cigarettes/pack from 20 to 19, and increased prices of recently introduced low-priced brands. Light cigarettes registered their lowest market share of the last 11 years (21.3 percent), while full-bodied American blend cigarettes have shown substantial gains. Market reports from April and May indicate that Germany's traditional brands have regained up to one-third the amount lost to the roll-your-own segment, which apparently peaked in early 1983 (coinciding with a price war between traditional cigarette manufacturers). While tobacco tax changes are not anticipated, all cigarette packages and marketing material will carry health warnings beginning January 1, 1984. High German cigarette prices have aided business in Luxembourg, France, Belgium, the Netherlands, and Switzerland. Also discussed are domestic cigarette consumption trends in the Netherlands, Switzerland, and Austria.

84–0333. Young, T. **The Demand for Cigarettes: Alternative Specifications of Fujii's Model.** *Applied Economics* 15(2): 203-211, April 1983.

Results indicate that the market for cigarettes is characterized by asymmetry of consumer response to changes in price and income levels, consistent with the hypothesis of consumer addiction. Two types of response are modeled: (1) formation of habits as income rises, regardless of product price; and (2) formation of habits when prices are low that are not readily given up as prices rise. Adaptation to a price or income change is gradual, but movement along the demand curve is irreversible when consumption habits have developed. Thus, asymmetry in consumer response is not just a shortrun phenomenon. The ratchet models suggest that the shortrun elasticity to price rises is around -.03, whereas responses to reductions in income would be about 0.15, indicating more inelastic responses than those suggested by the symmetric equation. It is concluded that policy measures to reduce consumption by manipulating market forces would need to be more severe than would be suggested by the demand elasticities calculated from the symmetric demand equation. In particular, where it has been suggested that a price rise of 10 percent (induced by a change in taxation) would lower consumption by 4.5 percent, the ratchet specifications of the demand equation indicate that a price rise of 14 percent would be required to obtain the same reduction in consumption. Hence, taxation policy would seem to be somewhat less powerful than has been suggested previously. (Auth. Abs. Mod.)

LEGISLATION

84–0334. Goerth, C. R. **Clearing the Smoke Through Law, Courts.** *Occupational Health and Safety* 52(9): 54, 56, September 1983.

Antismoking forces have taken action on the legislative front. Statutes regulating smoking, passed in six States, either prohibit smoking in public areas (i.e., banks, schools, public buildings, stores, restaurants, and bowling alleys) or require owners to designate specific smoking areas. San Francisco's antismoking ordinance requires all city business establishments to reasonably accommodate nonsmokers. Results of labor arbitration cases indicate that employers may establish smoking and nonsmoking areas of reasonable location and size. A complete ban against smoking must be supported by a rationale, such as equipment damage, fire hazard, procedural interference (i.e., lab tests), or reduction of productivity. Also discussed are recent and current legal action strategies and lawsuits by antismoking forces; smoking-related regulations; and the counterattack by advocates of smoking.

84–0335. Rizio, D. **US Industry Fights Self-Estinguishing Cigarette Bills.** *Tabak Journal International* 4: 316, 318, August 1983.

The proposed H.R. 1880, The Cigarette Safety Act, pending on the national level, would require cigarettes to meet performance standards for self-extinguishing. Additionally, a bill on the same topic was brought to the Senate, and, by early spring of this year, at least nine States had introduced 11 similar bills. At the time of writing, none of the bills had been passed. However, a lengthy hearing was held late in March regarding The Cigarette Safety Act. Supporters of the legislation argue that cigarettes are the leading cause of fire-related deaths and injuries. The tobacco industry, however, described the legislation as "a quick-fix solution" to a small part of the fire problem in the United States.

GENERAL

84–0336. Anderson, I. **Low-Tar Cigarette Is a Myth.** [illegible] *Scientist* 99(1368): 252, July 28, 1983.

Smokers have learned how to manipulate the engineering devices built into cigarettes to make them low-yield. By blocking ventilation holes or changing puffing patterns, smokers extract as much nicotine as desired from the low-yield cigarettes. It is noted that a smoker's blood cotinine level correlates with the number of cigarettes consumed, but not with the yield of the cigarette. Because smoking machines, which determine the values indicated on cigarette packages, do not smoke the way people do, these numbers are claimed to be misleading. A tobacco industry spokesman states that no claims as to safe or unsafe levels were ever made, and that complaints concerning government tar and nicotine yields should be addressed to the government.

84–0337. Bayerisches Staatsministerium des Innern. **Raucher-Report. [Report on Smokers.]** Bayerisches Staatsministerium des Innern RB-Nr. 03A/82/06, January 1983, 80 pp., German.

A report on smokers is provided by the Bavarian Ministry of the Interior. Topics discussed are (1) incidence of smoking; (2) marketing and advertising; (3) motivation, factors, smoking conditions; (4) health damage; (5) passive smoking; (6) light cigarettes; (7) smoking cessation methods; (8) information and education on drugs; (9) media; and (10) suggested reading. A list of 136 references is provided.

84–0338. Brotherston, J. **Tenth Birthday of ASH.** *Royal College of Physicians of Edinburgh* 13(2): 13-15, May 1983.

In the 10 years following the formation of the Scottish Committee of Action on Smoking and Health (ASH), smoking among Scottish males fell from 53 percent in 1972 to 46 percent in 1980, but the percentage of women smokers has remained static at 42 percent. More importantly, public attitudes have changed radically: the nonsmoker is now in the majority, meetings and social occasions are increasingly smoke-free, and cigarette use is becoming a dirty habit that should be abandoned if possible. Although ASH Scotland cannot claim credit for all progress, its activities have done much to accelerate change. A few of these are the recently published *The Scottish Epidemic* and 13 fact sheets; establishment of the smoking withdrawal research group; constant vigilance in the media; and numerous articles, workshops, exhibitions, and educational activities. Scotland currently is at the top of the international league list in number of fatalities from smoking-related diseases.

84–0339. Harriman, E. **Do Cigarettes Cheat the Tar Tables?** *New Scientist* 99(1366): 85-87, July 14, 1983.

In response to controversy in the United States over the Barclay "Actron" filter, the U.K. Department of Health and Social Security (DHSS) is questioning both the accuracy of its tar tables and its policy of encouraging smokers to switch to low-tar cigarettes. While smoke yield information is published to allow smokers to make informed decisions about the brands they smoke, machines do not smoke the way people do, and variations in puffing and blocking of ventilation holes can increase yields by 59 to 293 percent. In the past, the Independent Scientific Committee on Smoking and Health has worked closely with the tobacco industry on additives, substitutes, and "safer" cigarettes. As a result, tobacco sales have increased from 1,941 to 5,937 million pounds since 1973, and 80 percent of cigarettes consumed have filters. It is also suggested that much of the drop in cigarette tar yields over the past several years is a result of measurement manipulations by the tobacco industry, or simply filters that "beat the machine." Although most major tobacco companies have machines that can be programed to smoke as people do, neither the DHSS nor the Government Chemist has such a machine. The Barclay filter controversy, and related allegations by the U.S. Federal Trade Commission, are explored.

84–0340. Hadley, J. **More Medical Care, Better Health?** Washington, D. C., Urban Institute Press, URI 32900, 1982, 245 pp.

It is concluded that increased use of medical care does result in significantly lower mortality rates, although a reduction in smoking and/or other "health behavior" alterations would be more efficient. Smoking was included in an analysis of the processes that determine health levels and the economic theory of production. Using cross-sectional data for 1970, a series of health production functions were estimated, and an attempt was made to draw the connection between health-improving policies and the costs of inputs. The material is organized into nine chapters: (1) health and public policy; (2) the theory of health and health production; (3) variations in mortality rates (selected literature review); (4) defining a health indicator and measuring medical care use; (5) health production function estimates of adult cohorts; (6) health production function estimates of infant cohorts; (7) income, education, and mortality rates; (8) implications for policy; and (9) summary and conclusions.

84–0341. Jacobson, B. **Smoking and Health: A New Generation of Campaigners.** *British Medical Journal* 287(6390): 483-484, August 13, 1983.

The Fifth World Conference on Smoking and Health in Winnipeg differed from previous conferences, since participants recognized that the antismoking campaign is now a political battle to be waged against the tobacco industry itself. This was contrary to former ideas of smoking as mostly a medical problem to be solved by curing smokers of their "disease." Topics focused on three issues: the Third World, women, and children. Due to declining sales, the industry is investing in untapped markets among developing countries (now consuming 52 percent of all tobacco) and women. Tobacco in the Third World, however, not only kills people but also destroys the environment: 12 percent of trees cut down are used for tobacco curing, and tobacco production occupies land that could grow food. While emancipation has been blamed for the female smoking epidemic, it is noted that emancipated women are actually the least likely to smoke. A more obvious explanation may be the tobacco industry's deliberate marketing strategies aimed at women. Activities of some antismoking proponents that expose the industry's intentions are highlighted.

84–0342. Lenfant, C. **Are "Low-Yield" Cigarettes Really Safer?** *New England Journal of Medicine* 309(3): 181-182, July 21, 1983.

It has been shown that smokers generally respond to low-yield cigarettes by smoking differently or smoking more, to obtain the desired level of nicotine. Data indicate that smokers of low-yield cigarettes do not have a lower risk of myocardial infarction, coronary disease, or lung function decline than consumers of higher-yield cigarettes. Also, although those smoking the lower-yield products may have slightly less risk of lung cancer, their incidence rates are still higher than those of nonsmokers. The tar and nicotine delivery of a cigarette, as measured by a smoking machine, is not an accurate indication of exposure to the smoker because the machines do not smoke the way people do. Thus, there really is no less hazardous, or safe, cigarette.

84-0343. Medical Journal of Zambia. **The Smoking Epidemic.** *Medical Journal of Zambia* 14(3): 38-39, April-May 1980.

It is feared that smoking-related diseases will appear in developing countries before communicable diseases and malnutrition are controlled, increasing the strain on health services and widening the gap between the rich and poor countries. Zambia produces about four million kgs of tobacco yearly, with one-third consumed domestically (110 million cigarettes manufactured yearly, and 4.4 million kwacha/month spent on cigarettes). A recent survey at the University teaching hospital at Lusaka showed that a third of the academic staff and medical students were smokers, and half of the male workers with a primary school education also smoked. The importance of learning from the mistakes of industrialized countries, and the responsibility of health professionals in promoting health and discouraging dangerous habits is stressed. A brief review of smoking's association with overall mortality, cancer, cardiovascular disease, and reduced fetal growth, as well as the hazards of passive smoking, is provided.

84-0344. Moore, F. D.; Sieverts, S.; Stokes, J., III. **Why Not Rate Health and Life Insurance by Risks? (Letters).** *New England Journal of Medicine* 309(4): 246-247, July 28, 1983.

While a fellow physician enthusiastically supports an earlier proposal to modify health insurance rates to reflect avoidable health risks such as smoking (*New England Journal of Medicine* 308(7): 393-395, February 17, 1983) a spokesman for Blue Cross/Blue Shield expresses doubts as to whether such a system is feasible. Problems in identifying individuals with high-risk habits, setting rates, or just agreeing on the best targets to save health dollars would be compounded by complex recordkeeping, claims administration, diagnostic testing, billing, and collection. Family and group coverage would also complicate the premium determination. The avoidable risk of smoking presents special problems (the heavy smoker who quit 10 or 20 years ago; the risk difference between someone who smokes only one cigarette/day and the one who smokes 60/day). The threat of financial penalties would probably not be enough to persuade people to change their lifestyles. The original author believes that there are practical answers to the questions raised, but suggests a different forum for discussion. The main point of his argument (which was missed) is that fear and threats should be replaced, offering something in return for people who modify their behavior. Premium reductions and cash rebates are merely ways the insurance industry could cooperate. See also 83-1728.

84-0345. Rosen, R. A. **Cost of Smoking. (Letter).** *New England Journal of Medicine* 309(15): 929, October 13, 1983.

In response to a letter (*New England Journal of Medicine* 308(18): 1105, May 5, 1983) suggesting that health costs of smoking be shifted to the consumer, it is proposed that an assigned-risk system be used. This system, similar to that used in the automobile insurance industry, could use direct taxation of cigarette manufacturers to provide funds for health care of those with smoking-related diseases. The manufacturers' costs would be passed on to the consumer, which might even result in lowered consumption. If successful, the process could be applied to alcohol-related costs as well. See also 83-2066.

84-0346. Rublee, D. A. **Cigarette Sales in Delaware Hospitals. (Letter).** *Delaware Medical Journal* 55(8): 475, August 1983.

Although a nationwide survey (1982) found that cigarettes were being sold in only 30 percent of hospitals compared with 56 percent in 1979, 50 percent of Delaware hospitals are still selling cigarettes. Since there are few, if any, compelling medical reasons to permit either the sale or smoking of cigarettes in hospitals, the medical staffs should recommend to the governing boards that sales be eliminated as soon as possible. Smoking is unhealthful, leads to or is implicated in a number of disease processes, and has been labeled as the single greatest cause of premature death and disability in the United States. Because hospitals are increasingly being seen as sites for health promotion and education, it seems appropriate that a nonsmoking atmosphere be maintained.

84-0347. Shorten, E. **The Nurses Responsibility on the Ward.** *World of Irish Nursing* 12(1/2): 4, 6, January-February 1983.

The prime responsibilities of hospital nurses are quality of care delivered and education of self, patients, and visitors. Smoking-related health education must begin at the student nurse level and extend to the administrative and teaching staffs, and to nurses on the ward. Suggested approaches are (1) education by example, (2) hospital no smoking policies and areas, (3) separating smoking and nonsmoking patients, (4) enforcing smoking-related hospital rules, and (5) prohibiting cigarette sales on hospital premises. Nurses have the greatest responsibility in the prenatal, cardiac, thoracic, and gynecological wards. Job-related stress is cited as a smoking factor. Also discussed are health consequences of smoking, including cardiovascular, thoracic, passive smoking effects (i.e., a dose—response relationship between lung cancer and smoke exposure in nonsmoking Japanese wives), fetal effects (low birth weight, child development), and early menopause; and the interaction between smoking and metabolism of medications, proteins, carbohydrates, and vitamins.

84–0348. Silverstein, M.; Pak, R.; Maizlish, N. **Tobacco and Asbestos: Consistent Treatment Advocated.** *Journal of Occupational Medicine* 25(9): 694-695, September 1983.

The commendable restriction of smoking by employees in the asbestos industry does not reduce the risk of asbestos exposure, which has been shown to act synergistically with tobacco in disease etiology. The uncompromising stand on smoking is in sharp contrast with persistent efforts to resist more stringent workplace asbestos exposure standards. Consistency would require that the industry (1) allow workers to smoke reduced tar, filtered cigarettes (considered ridiculous); or (2) treat asbestos in the same absolute fashion and institute measures that would completely eliminate worker exposure. Because of the synergistic effects of smoking and occupational exposure to toxic substances such as asbestos, the National Institute on Occupational Safety and Health has recommended that research reflect both exposures. While many companies are now keeping records of employees' smoking habits, it has been all too easy to focus on workers instead of the workplace. It is hoped that studies assessing the interacting effects of multiple chemical workplace exposures, apart from the issue of smoking, will be developed.

84–0349. Tuomisto, J. **Medical Responsibility in Environmental Protection.** *Scandinavian Journal of Social Medicine* Supplement 31: 82-85, 1982.

In the total environment, no chemical or combustion product matches the importance of cigarette smoking as a health hazard. For example, smoking 1.4 cigarettes is comparable with consuming 100 charcoal-broiled steaks or living for 20 years near a polyvinyl chloride plant (exposures estimated to reduce life expectancy by 8 minutes). Medical responsibility in environmental protection involves four tasks: risk assessment, basic research, prediction of hazards in new activities, and education of the public. Sensitive short-term tests make it likely that hundreds or thousands of chemicals will be suspected of carcinogenic activity. The zero-tolerance concept is not feasible, and thus determination of acceptable levels of risk is an important responsibility. However, in public policy decisions, incomprehensibly low levels of potential carcinogens should not outweigh smoking and other risks that may be millionfold.

AGRICULTURE

84–0350. Gaines, T. P.; Csinos, A. S.; Stephenson, M. G. **Grade Index and Yield Correlations With Chemical Quality Characteristics of Flue-Cured Tobacco.** *Tobacco International* 185(17): 41-45, August 19, 1983.

Nicotine (Nic), total nitrogen (TN), and reducing sugar (RS) concentrations are associated with the chemical quality of flue-cured tobacco (*Nicotiana tabacum* L) and are used as criteria for new cultivars to be eligible for release. The effects of precipitation and temperature during the growing season were investigated for Nic, TN, and RS concentrations in flue-cured tobacco and on their relationships to crop quality and yield. The tobacco used was from the Georgia Official Tobacco Variety Test conducted at Tifton, Georgia, from 1975 through 1980. Weather affected concentrations of Nic, TN, RS, and grade and yield during the 6-year study. Grades were high during the 3 years the weather was favorable for good tobacco production, and low in the 3 years the weather was atypically wet or dry and hot. A balance in chemical constituents correlated better with grade and yield than chemical constituents compared individually. Grade and yield were correlated positively with the TN/Nic ratio and the RS/Nic ratio. A negative correlation was found between nicotine concentration and grade and yield. McNair 944 and NC 79 had the more favorable yield, grade, and chemical quality characteristics of the cultivars evaluated in the crop performance tests. (Auth. Abs.)

84–0351. Lorenz, W.; Bahadir, M.; Korte, F.; Drawert, F. **Pestizide im Tabakrauch. II. Pyrolyseverhalten von Ausgewahlten Pestiziden Beim Tabakrauchen. [Pesticides in Tobacco Smoke. II. Pyrolytic Behavior of Certain Pesticides During Tobacco Smoking.]** *Chemosphere* 12(2): 271-275, 1983, German.

The fate of lindane, DDT, malathion, monolinuron, captan, and maneb during the smoking process was studied. It was observed that the amount of unchanged pesticides in the mainstream smoke depends on steam volatility of the substances, while the vapor pressures have no significant influence. Distribution of partial fractions in the mainstream smoke, sidestream smoke, filters, and butts, was relatively uniform for all the pesticides considered. However, considerable differences were found in the transfer of nondecomposed compounds into mainstream smoke and in formation of $^{14}CO_2$ in sidestream smoke. Analysis showed that a filter unselectively eliminated about half of the pesticides in the mainstream smoke. (Auth. Abs. Mod.)

CHEMISTRY

84–0352. Awotedu, A. A.; Higenbottam, T. W.; Onadeko, B. O. **Tar, Nicotine, and Carbon Monoxide Yields of Some Nigerian Cigarettes.** *Journal of Epidemiology and Community Health* 37(3): 218-220, September 1983.

None of the 14 brands of cigarettes manufactured and commonly available in Nigeria that were analyzed for tar, nicotine, and carbon monoxide content were in the low-tar (<17 mg/cigarette) category. Five were in the middle- to high-tar category (>22 mg/cigarette) and nine were in the middle-tar category (17 to 22 mg/cigarette). The nicotine and carbon monoxide levels were similar to those of European cigarettes. The only available data on yields from cigarettes manufactured in Africa showed a disparity in tar yields when compared with similar brands sold in the United Kingdom; one brand in Kenya contained 31 mg tar and 2.0 mg nicotine while the same brand in the United Kingdom had only 18 mg tar and 0.9 mg nicotine. While recent attempts to enact legislation to regulate cigarette promotions and require health warnings have been rejected due to lack of data, it is hoped that the information now available will encourage such legislation. Tobacco companies need to manufacture low-tar cigarettes in developing countries as is the practice in the developed parts of the world. (Auth. Abs. Mod.)

84–0353. Baker, R. R. **Formation of Carbon Oxides During Tobacco Combustion. Pyrolysis Studies in the Presence of Isotopic Gases to Elucidate Reaction Sequence.** *Journal of Analytical and Applied Pyrolysis* 4(4): 297-334, March 1983.

A sequence of general chemical steps for the production of carbon oxides and water during tobacco combustion is described, based on study data and previously published studies on graphite and coal reactions. During the combustion of tobacco, carbon monoxide is formed by the thermal decomposition of tobacco, by reaction of tobacco with atmospheric oxygen, and by secondary reaction of tobacco with primary products such as carbon dioxide and water. These three processes occur in parallel and are interdependent. The temperature ranges over which each process occurs, and their relative importance, were assessed by pyrolyzing tobacco in the presence of various isotopically labeled gases. Nonisothermal pyrolyses were conducted at a heating rate of $1.6\ K\ s^{-1}$ up to 1000°C, with the products analyzed by mass spectrometer. Pyrolysis in the presence of oxygen-18 indicates that combustion of tobacco starts at 180°C. Carbon dioxide and water are formed by combustion at 180°C, while carbon monoxide is not formed as a combustion product until 460°C. The quantities of carbon monoxide and dioxide formed by thermal decomposition of tobacco above 400°C are significantly reduced by the occurrence of combustion. Pyrolysis in the presence of carbon-13 dioxide or carbon dioxide-18 shows that its major reaction, endothermic reduction to form carbon monoxide, begins at 450°C. Pyrolysis in an oxygen-18/carbon-13 dioxide atmosphere has shown that this endothermic reduction of carbon dioxide occurs in parallel with the strongly exothermic oxidizing reactions. Thirty percent of the total carbon monoxide formed was produced by thermal decomposition of the tobacco, 36 percent by combustion of the tobacco, and at least 23 percent via carbon dioxide. The remainder was produced by an interaction of carbon dioxide reduction and oxidation. Similar proportions would be expected inside the reaction zone of a burning cigarette. Pyrolysis in the presence of heavy water has shown that the major reaction of the water is to quantitatively produce carbon monoxide and hydrogen above 600°C. Considerable isotopic exchange reactions also occur. Pyrolysis in the presence of carbon monoxide-18 has shown that carbon monoxide reacts with tobacco to a small extent at temperatures above 220°C, mainly to abstract oxygen combined in the tobacco and produce carbon dioxide. (Auth. Abs. Mod.)

84–0354. Bokelman, G. H.; Ryan, W. S., Jr.; Oakley, E. T. **Fractionation of Bright Tobacco.** *Journal of Agricultural and Food Chemistry* 31: 897-901, July-August 1983.

Results of research to develop methods for the systematic fractionation and characterization of cell wall biopolymers in tobacco, based on procedures that had been used with other plant materials, are described. Polymeric cell wall components were determined for ground, uncased bright tobacco lamina by using a systematic fractionation scheme. Starch was determined by an enzymatic procedure utilizing a thermophilic amylase. Analysis for pectin utilized an acid-catalyzed decarboxylation method. Lignin was determined by both the Klason lignin procedure and ^{13}C NMR integration of aromatic carbons. Estimation of protein was based on the results of Kjeldahl digestion. Hemicellulose and cellulose were determined by a capillary gas chromatography procedure for quantitation of neutral sugars. Results were compared with those from a serial extraction procedure. Values for all components, except cellulose, were found to vary considerably between the two procedures. Errors in the serial extraction procedure were attributed to the fact that many distinct types of biopolymers have overlapping ranges of solubility. The cell walls of cured bright lamina were found to resemble the primary cell walls of dicots. (Auth. Abs. Mod.)

84–0355. Bolt, A.J.N.; Purkis, S. W.; Sadd, J. S. **A Damascone Derivative From** ***Nicotiana Tabacum.*** *Phytochemistry* 22(2): 613-614, 1983.

A new member of the damascone series, 4-hydroxy-β-damascone, was identified in the steam-distillable oil from Virginia tobacco. The carbon skeleton of 4-hydroxy-β-damascone was established by chemical oxidation to a known compound, a component of the aged β-damascone mixture characterized using existing physicochemical data. Dehydration of 4-hydroxy-β-damascone gave damascenone. Accurate mass measurement of the molecular ion gave the molecular formula as $C_{13}H_{20}O_2$. The IR spectrum indicated hydroxyl and carbonyl groups, the position of the latter absorption being typical of a conjugated unsaturated ketone. Other fragments of the structure suggested by the IR spectrum were a gem-dimethyl group and a conjugated *trans*-disubstituted olefin. The ^1H NMR spectrum showed a typical ABX_3-type coupling with chemical shifts and coupling constants consistent with a *trans*-crotonyl fragment. This was also strongly indicated by the mass spectrum with ions at m/z 69, 41, and 139 $(M—69)^+$. The absence of any further olefinic protons fixed the position of the second double bond at C-5. The position of the hydroxyl group was indicated by the multiplicity of the NMR absorption of the methine proton and confirmed by oxidation of 4-hydroxy-β-damascone to a known compound. (Auth. Abs. Mod.)

84–0356. Brunnemann, K. D.; Scott, J. C.; Hoffmann, D. *N*-Nitrosoproline, an Indicator for N-Nitrosation of Amines in Processed Tobacco. *Journal of Agricultural and Food Chemistry* 31(4): 905-909, July-August 1983.

Tobacco and tobacco smoke contain three types of *N*-nitrosamines (all known carcinogens) that are formed during processing. The goal of this study was to explore the possible presence of *N*-nitrosoproline (NPRO) in tobacco and tobacco smoke and to investigate whether this compound could serve as an indicator for the formation of tobacco-specific *N*-nitrosamines (TSNA) during tobacco processing and smoking. The advantage of measuring N-nitrosation potential by determining the noncarcinogenic NPRO lies in avoiding the carcinogenic TSNA for which reference compounds are not always readily available. NPRO was isolated from processed tobacco, and a method for its quantitative assessment was developed, based on the enrichment of NPRO by solvent distributions, derivation to its methyl ester, and gas chromatography with a thermal energy analyzer. Cigarette and cigar tobaccos contained 0.33-2.3 ppm of NPRO; chewing tobacco and snuff had levels of 0.45-21.8 ppm. On the basis of the analyses of 14 tobacco products, the formation of the noncarcinogenic NPRO is significantly correlated with the formation of *N'*-nitrosonornicotine and the sum of the carcinogenic TSNA. Fine-cut snuff is relatively rich in NPRO and TSNA. The latter, the only known carcinogens in snuff tobaccos, occur in relatively high concentrations. It is suggested that efforts be undertaken to inhibit *N*-nitrosamine formation, especially during the preparation of fine-cut snuff. NPRO was not detected in cigarette smoke (<1 ng/cigarette). (Auth. Abs. Mod.)

84–0357. Chamberlain, W. J.; Arrendale, R. F. **An Alternate Method for the Analysis of** ***N*****-Nitrosonornicotine in Tobacco.**

Journal of Agricultural and Food Chemistry 31(4): 909-911, July-August 1983.

Flue-cured tobacco samples differing in nicotine and nornicotine content were analyzed by modified gas chromatography with a nitrogen-phosphorus detector to determine if a positive relationship exists between nicotine and nornicotine levels and *N*-nitrosonornicotine (NNN). Results indicate that percentages of nicotine, nornicotine, total alkaloids, and nornicotine/nicotine ratios were not associated with NNN levels in "normal" tobaccos, in which nicotine constituted approximately 92 percent of the total alkaloids. In "converter" tobaccos, which appear to convert nicotine to nornicotine and thus have significantly higher ratios of nornicotine to nicotine, there also appeared to be no correlation of nornicotine levels with NNN levels. (Auth. Abs. Mod.)

84-0358. Hill, D.; Brande, W.; Phillips, D.; Pomerleau, O. **A Rapid Plasma Extraction Method for Gas Chromatographic Analysis of Nicotine.** *Analytical Letters* 16(B5): 355-365, 1983.

A method for nicotine extraction and analysis from human plasma, using thermionic detection, is described. The procedure provides approximately 100 percent accuracy at nicotine levels above 5 ng/ml. Between-day reproducibility was satisfactory, as evidenced by a 4.4 percent coefficient of variation for a 20 ng/ml sample analyzed over a 3-day period. Concentration of the sample without evaporation required less than 1 hour for preparation of 20 samples.

84-0359. Liu, D.; Sheen, S. J. **Observations of Variability in Calli and Regenerated Plants From a Cell Culture of Samsun NN Tobacco.** *Tobacco International* 185(20): 71-73, September 30, 1983.

Morphological, physiological, and virus-resistant characteristics of calli or plants regenerated from cell cultures of Samsun NN tobacco varied considerably. Morphological variations included color, growth, and differentiation rate of calli; leaf shape; and growth pattern of regenerated plants. The amount of alkaloids and polyphenols was significantly larger in leaves of regenerated plants than Samsun NN; however, Samsun NN had a higher concentration of total nitrogen and a comparable or smaller level of soluble sugar. Electrophoretic patterns of isoperoxidases from the leaf extract of regenerated plants differed for the cathodic and slow-moving anodic variants, but not for the fast-moving anodic ones. All regenerated plants exhibited tobacco mosaic virus resistance but varied from no spots to numerous, large necrotic spots. (Auth. Abs.)

84-0360. Lorenz, W.; Bahadir, M.; Korte, F.; Drawert, F. **Pestizide im Tabakrauch. I. Methode zur Bestimmung des Uberganges von Pestiziden und Deren Pyrolyseprodukten in Haupt- und Nebenstromrauch. [Pesticides in Tobacco Smoke. I. Method to Determine the Transfer of Pesticides and Their Pyrolysis Products Into the Main- and Sidestream Smoke.]** *Chemosphere* 12(2): 263-269, 1983, German.

A smoking machine that simultaneously absorbs pyrolysis products from sidestream and mainstream smoke is described. The apparatus allows analysis of the transfer of pesticides and their pyrolysis byproducts into mainstream and sidestream smoke, as well as their absorption on filters and cigarette butts. The system was tested with experimental cigarettes spiked with 4,4'-DDT, DDD, DDE, and DDM, and bis-(4-chlorphenyl)-methane. Results were compared with animal studies.

84-0361. Mishra, U. C.; Shaikh, G. N. **Determination of Trace Element Concentrations of Indian Cigarette Tobacco by Instrumental Neutron Activation Analysis.** *Journal of Radioanalytical Chemistry* 78(2): 385-390, 1983.

No significant differences in trace element concentration between cigarette brands or among forms of Indian tobacco were found by instrumental neutron activation analysis. Five brands of Indian cigarette tobacco, two of chewing tobacco, and two of bidi tobacco were analyzed for 12 trace elements, and levels compared with those in American, German, Iranian, and New Zealand tobaccos. Iron and zinc levels were higher in Indian than in German and Iranian tobaccos. Among toxic metals, only zinc, cobalt, and chromium levels were higher in Indian than in American tobaccos. It is concluded that the smoking hazard does not depend on either the relative cost of the tobacco product nor on its origin.

84-0362. Oakley, E. T. **Enzymatic Determination of Starch in Fresh Green, Lyophilized Green, and Cured Tobacco.** *Journal of Agricultural and Food Chemistry* 31(4): 902-905, July-August 1983.

An enzymatic method for the determination of starch in cured, fresh green, and lyophilized green tobaccos is described. The naturally present sugars are extracted with an aqueous ethanol distillate, and the starch solubilized in boiling buffer solution. The starch is converted to glucose enzymatically, and the glucose is determined colorimetrically by a manual glucose oxidase method. Quantitative data are presented for starch in cured bright tobacco and in fresh and freeze-dried green bright tobaccos. Comparison is made of starch values obtained by a colorimetric method based on the starch-iodine complex. Accuracy of the method was established by repetitive extraction of residual tobacco after the completion of the procedure. Relative standard deviations at the 2σ level are ±12 percent on cured bright tobacco with 3.2 percent starch, ±14 percent on fresh, green tobacco with 15.2 percent starch, and ±3 percent on lyophilized green tobacco with 15.4 percent starch.

84-0363. Parrish, M. E.; Good, B. W.; Jeltema, M. A.; Hsu, F. S. **Pattern Recognition and Capillary Gas Chromatography in the Analysis of the Organic Gas Phase of Cigarette Smoke.** *Analytica Chimica Acta* 150(1): 163-170, June 1, 1983.

Distinguishing between several cigarette types simultaneously by glass capillary gas chromatography requires the use of multivariate statistics for data reduction, pattern extraction, and ranking the importance of the chromatographic peaks. These techniques were applied to the gas phase of the cigarette smoke. Ten cigarettes were studied, both cased and uncased, of 100 percent Bright, 100 percent Burley, 100 percent Oriental tobacco, and blends of 33/33/33 percent and 60/30/10 percent, respectively. At least five chromatographic profiles were obtained for each different cigarette, giving a total of 51 chromatograms to serve as the data base. From each chromatogram, containing about 100 peaks, a subset of 20 peaks was selected manually. The data were evaluated by using discriminant analysis and factor analysis. The former technique produced a satisfactory separation of cigarette types into distinct groups, and results from the factor technique were successful in providing discrimination based on three factors. (Auth. Abs.)

84–0364. Perfetti, T. A. **Structural Study of Nicotine Salts.** *Beitraege zur Tabakforschung International* 12(2): 43-54, June 1983.

Salt formation between organic acids and nicotine is dependent upon the structure of the acids (aliphatic or aromatic) and their functionality. The structures of three types of nicotine salts, with acid to base ratios of either 1:1, 2:1, or 3:1 were determined through infrared, ultraviolet, proton nuclear magnetic resonance, and both carbon nuclear magnetic resonance and field desorption—mass spectroscopy. Results showed that the 1:1 salts of nicotine have amino acids or benzoic-type acids bound to the *N*-methylpyrrolidine nitrogen of nicotine. The 2:1 salts are found to bind to one acid group as in the 1:1 salts and a second to the nitrogen of the pyridine ring. The 2:1 salts of nicotine are formed with formic acid, aliphatic dicarboxylic acids, and/or nitroaromatic acids. Nicotine forms 3:1 salts with aliphatic monocarboxylic acids starting with acetic acid. One acid is bound as in the 1:1 salts, while the other two acids dimerize and bind to the nitrogen of the pyridine group. Tables list the infrared absorption bands and physical characteristics of nicotine salts; carbon nuclear magnetic resonance shifts of nicotine, pyrrolidine, and pyridine salts; and proton nuclear magnetic resonance on chemical shifts of nicotine salts. (Auth. Abs. Mod.)

84–0365. Rickert, W. S.; Robinson, J. C.; Collishaw, N.E.; Bray, D.F. **Estimating the Hazards of "Less Hazardous" Cigarettes. III. A Study of the Effect of Various Smoking Conditions on Yields of Hydrogen Cyanide and Cigarette Tar.** *Journal of Toxicology and Environmental Health* 12(1): 39-54, July 1983.

A cigarette brand with nominal tar and nicotine yields (4.0 mg and 0.4 mg, respectively) was examined under various machine-smoked conditions reflecting the wide range of human smoking behavior. Three levels of each of five smoking parameters (butt length, puff duration, puff interval, puff volume, and ventilation occlusion) were examined. The effects on puff number and total particulate matter (TPM) as well as gas phase, particulate phase, and total hydrogen cyanide (HCN) yields were estimated. Yields of HCN (a ciliatoxic agent) and TPM varied significantly under different smoking conditions. Ventilation occlusion had the most pronounced effect, accounting for 34 percent of the response variation of TPM yields and 42 percent of the response variation for total HCN yields. Testing of 115 brands produced total HCN yields from 2 to 233 μg/cigarette (a range less than that observed for a single brand smoked under various nonstandard conditions), providing a possible explanation for the previously noted lack of correspondence between HCN yields under standard conditions and levels of thiocyanate in samples of smokers' plasma and saliva. In addition, HCN yields and efficiency of filters in removing HCN were examined under standard smoking conditions. Acetate filters, the most common variety, were found to remove an average of about 14 percent of the HCN from mainstream smoke. (Auth. Abs.)

84–0366. Siddiqui, I. R.; Rosa, N. **Low Molecular Weight Carbohydrates of Tobacco.** *Tobacco International* 185(21): 27-31, October 14, 1983.

Fourteen low molecular weight carbohydrates from the 80 percent ethanol-soluble fraction of Bright tobacco (*Nicotiana tabacum*, Delhi 76) were characterized either in the crystalline form or by the isolation of crystalline derivatives; or by the results of partial hydrolysis and paper chromatography, paper electrophoresis, gas liquid chromatography (GLC), and GLC with M-sulphuric acid of their trimethylsilyl derivatives. The average percentage composition in the 80 percent ethanol-soluble fraction (36.5 percent) of cured lamina was D-ribose, 0.11; D-xylose, 0.19; D-fructose containing 0.97 percent D-psicose, 9.61; D-glucose, 3.24; D-sorbitol, 0.02; myo-inositol, 1.04; 1-deoxy-1-((S)-2-(3-pyridyl)-1-pyrrolidinyl)β-D-fructopyranose, 0.26; sucrose, 6.24; maltose, 0.34; gentiobiose, 0.01; D-glucopyranosyl myo-insitol, 0.04; erlose, 0.02; and theanderose, 0.01. Besides malic acid (butanedioic acid), 11.02 percent, three nonreducing acidic components (T 0.23, 0.31, and 0.47) amounted to 0.74 percent, 0.09 percent, and 0.34 percent, respectively. Among the unknown, three components present in significant amounts (1.23, 0.09, 1.11 percent) showed relative retention time values of 0.25, 0.267, and 0.56, respectively. (Auth. Abs.)

84–0367. St. Aubyn, A.W.M. **No Rigging of Tar Tests. (Letter).** *New Scientist* 99(1373): 642, September 1, 1983.

Statements and observational methods discussed in a previous article (*New Scientist* 99(1370): 431, August 11, 1983), which claimed that automated cigarette machines are rigged to underestimate tar and nicotine deliveries under human conditions, are disputed. It is asserted that a smoking machine is designed to objectively estimate tar and nicotine yields for comparative purposes and therefore would not be expected to mimic every smoker's habits. Furthermore, the puff volume, duration, and frequency, and other constants in the standard U.K. methodology for smoking cigarettes by machine to establish tar yield, were established in properly conducted, fully representative observational studies and are accepted by the government, tobacco industry, and the Independent Scientific Committee on Smoking and Health. See also 84-0587

84–0368. Wilson, R. A.; Mookherjee, B. D.; Vinals, J. F. **The Volatile Constituents of Virginia, Burley, Turkish and Black Tobaccos.** *Tobacco Reporter* 110(10): 42-46, October 1983.

Comparative survey analyses of the total oils of Burley, Virginia, Turkish, and black Dominican tobaccos have yielded a "new generation" of tobacco flavors expected to meet the tobacco industry's present and future needs. Of 80 fractions analyzed, the smoke evaluations of fractions 21, 26, and 53-54 were considered the most significant. The major component of fraction 21 was solanone; two novel compounds, methylthiodamascenone and methylthio β-damascone, were identified in this fraction. Solanone and geranyl acetone accounted for more than 60 percent of the peak area of fraction 26, with damascenone and β-damascone also present. However, fractions 53 and 54, which contribute sweet, hay, tobacco-like, barnyard-like aromatics and very good body to tobacco smoke, were preferred over all others. Gas liquid chromatographic analysis showed a completely different pattern (with the exception of solanone), compared with fractions previously observed. All five isomers of the megastigmatrienones were present in major amounts, as well as dihydrooxoedulan, solanofuran, and significant quantities of tetrahydropseudodamascone, solavetivone, 4-methylthio-2-butanone, α-homocyclogeraniol, and dihydrobovalide. It is indicated that this may be the first report of both tetrahydropseudo-damascone and α-homocyclogeraniol in tobacco. The analytic procedures used and potential routes to commercialization are described, and smoke evaluations for components of fraction 53-54 are provided.

PHARMACOLOGY AND TOXICOLOGY

84–0369. Abood, L. G.; Latham, W.; Grassi, S. **Isolation of a Nicotine Binding Site From Rat Brain by Affinity Chromatography.** *Proceedings of the National Academy of Sciences of the United States of America* 80(11): 3536-3539, June 1983.

With the use of affinity chromatography, a (^{3}H)-nicotine binding site was purified almost 1,000-fold from a Triton X-100-solubilized extract of rat brain neural membranes. The affinity column was prepared by conjugation of (*R,S*)-6-(2-hydroxyethyl)nicotine in epoxy-activated Sepharose. Further purification of the material from the affinity column was resolved by using another column of the same affinity gel, resulting in the isolation of a major protein (about 95 percent purity) that had a M_r of 56,000, as determined by NaDodSO$_4$/polyacrylamide gel electrophoresis, with very minor components ranging in M_r from 47,000 to 83,000. With the use of various nicotine analogs, it was shown that the purified material exhibited nearly identical binding characteristics to rat brain membrane preparations, including stereoselectivity for the nicotine enantiomers. The K_d of the purified site, 3.5x10^{-9}M, was similar to that observed with membrane and Triton X-100-soluble preparations, whereas the binding capacity was >25 pmol/mg of protein, compared with 0.07 pmol/mg of protein in the starting material. The results are discussed in relation to the purified nicotinic cholinergic receptor from electroplax. It is concluded that the nicotine site in rat brain is different from the cholinergic receptor of electroplax or calf skeletal muscle. (Auth. Abs.)

84–0370. Benowitz, N. L.; Kuyt, F.; Jacob, P., III; Jones, R. T.; Osman, A.-L. **Cotinine Disposition and Effects.** *Clinical Pharmacology and Therapeutics* 34(5): 604-611, November 1983.

Results indicate that, at levels to which cigarette smokers are generally exposed, cotinine (the main metabolite of nicotine in man), precipitates no cardiovascular activity, and weak, if any, psychologic activity. Cotinine disposition kinetics were studied in 28 healthy habitual cigarette smokers. Eight subjects received cotinine fumarate, 4 µg base/kg/min i.v. for 60 min. Mean metabolic clearance was 60±12 ml/min and mean renal clearance was 12±5 ml/min, averaging 17 percent of total clearance. Terminal t1/2 averaged 15.8±4.0 hour in the 8 subjects and 19.7±6.5 hour in 12 subjects who abstained from smoking for 3 days. The effect of urinary acidification and alkalinization on renal clearance of cotinine during cigarette smoking was studied in another group of eight subjects. Compared with baseline, renal clearance was increased about 50 percent by urinary acidification but it was not affected by alkalinization. Infusion of cotinine to blood concentrations seen in moderately heavy smokers had no effect on heart rate, blood pressure, or skin temperature, measures that are sensitive to the effects of nicotine. No spontaneous subjective effects were reported. Because of wide variations in clearance rates, it is suggested that in planning followup sampling in smoking cessation studies, at least 1 week of reported nonsmoking should pass before the blood concentration of cotinine is measured to assess smoking status. (Auth. Abs. Mod.)

84–0371. Boudier, C.; Pelletier, A.; Pauli, G.; Bieth, J. G. **The Functional Activity of α$_1$-Proteinase Inhibitor in Bronchoalveolar Lavage Fluids From Healthy Human Smokers and Non-**

Smokers. *Clinica Chimica Acta* 132(3): 309-315, August 31, 1983.

The percentage of functionally active α_1-proteinase inhibitor in bronchoalveolar lavage fluid was 54±12 in smokers and 38±14 percent in nonsmokers (nonsignificant difference). Data disagree with earlier reports suggesting the presence of fully active α_1-proteinase inhibitor in nonsmokers and of only partially active inhibitor in smokers. Cigarette smoking is an important risk factor for pulmonary emphysema. In vitro experiments document cigarette smoke-induced inactivation of α_1-proteinase inhibitor, a protein that is thought to protect the lung interstitium against the deleterious action of neutrophil elastase. To assess the relevance of this in in vitro findings, the functional activity of α_1-proteinase inhibitor collected by bronchoalveolar lavage from 20 healthy volunteers (10 heavy smokers and 10 nonsmokers) was measured. The total inhibitor concentration was measured by radial immunodiffusion. The active inhibitor concentration was determined by elastase inhibitory capacity. A pure and active site-titrated porcine pancreatic elastase and a kinetic assay with succinyl-trialanine-*p*-nitroanilide were used to provide reliable and accurate results. A possible explanation for the observed differences may be that smokers in this study abstained for 24 hours before lung lavage, whereas in at least one other study, subjects were permitted to smoke until the lavage procedure. (Auth. Abs. Mod.)

84–0372. Brischetto, C. S.; Connor, W. E.; Connor, S. L.; Matarazzo, J. D. **Plasma Lipid and Lipoprotein Profiles of Cigarette Smokers From Randomly Selected Families: Enhancement of Hyperlipidemia and Depression of High-Density Lipoprotein.** *American Journal of Cardiology* 52(7): 675-680, 840, October 1, 1983.

Cigarette smokers (61 males and 52 females) had significantly lower high density lipoprotein levels and higher very low density lipoprotein and plasma triglyceride levels than the 119 ex-smokers and 218 nonsmokers from 233 randomly selected American families. Cigarette smoking is known to be a risk factor for coronary disease, and it is hypothesized that it may be the result of effects on plasma lipids. Plasma level measurements showed that lipids and lipoproteins were related to the number of cigarettes smoked/day. Heavier cigarette smokers (>25 cigarettes/day) had significantly lower high density lipoprotein levels and significantly higher very low density lipoprotein total cholesterol and plasma triglyceride levels than those who smoked <25 cigarettes/day, nonsmokers, and ex-smokers. The lipid and lipoprotein values of those who smoked <15 cigarettes/day were similar to those of ex-smokers and nonsmokers. Since ex-smokers had levels of plasma lipids and lipoproteins similar to those of nonsmokers, the findings add another health-enhancing benefit to the cessation of smoking. A continuing Medical Education test on the subject matter included in the report is also provided. (Auth. Abs. Mod.)

84–0373. Brittebo, E. B.; Castonguay, A.; Furuya, K.; Hecht, S. S. **Metabolism of Tobacco-Specific Nitrosamines by Cultured Rat Nasal Mucosa.** *Cancer Research* 43(9): 4343-4348, September 1983.

The metabolism of two tobacco-specific nasal carcinogens, N'-nitrosonornicotine (NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), was investigated using cultured nasal septa of F344 rats. Tissue explants were cultured with ^{14}C-labeled *N*-nitrosamines, and unbound metabolites present in the medium were quantitated by high performance liquid chromatography. The mucosa of the nasal septum had a marked capacity to metabolize NNN and NNK to hydroxylated products that were released into the culture media. Extensive activation by α-carbon hydroxylation of NNN (preferentially 2′-carbon hydroxylation) and NNK was observed, whereas no deactivation by pyridine *N*-oxidation could be detected. Microautoradiographic studies of explants showed that binding of radioactivity occurred preferentially in the respiratory and olfactory epithelia and in the subepithelial glands of the nasal mucosa. It is suggested that reactive metabolites of NNN and NNK are formed within the target tissue rather than being transported from the liver to the nasal mucosa. Cultures of nasal septa can be used to ascertain the role of the nasal mucosa in the activation of nasal-specific carcinogens. (Auth. Abs.)

84–0374. Brown, R. C.; Fleming, G.T.A.; Knight, A. I. **Asbestos Affects the *In Vitro* Uptake and Detoxification of Aromatic Compounds.** *Environmental Health Perspectives* 51: 315-318, September 1983.

Findings confirmed that fibrous mineral dusts have more ability than nonfibrous dusts to catalyze the transformation of benzo(*a*)pyrene (BaP) from the microcrystalline state into lipid solution. Macromolecular binding metabolites of BaP were formed in A[illegible]9 cells to a similar extent whether BaP was added in solution or adsorbed to fibers; however, the level of water-soluble metabolites was lower in cultures treated with adsorbed hydrocarbon. It was found that asbestos can also inhibit the accumulation of 1-naphthyl glucuronide in cultures treated with 1-naphthol. The significance of this in asbestos pathogenesis is discussed. (Auth. Abs. Mod.)

84–0375. Chang, M.J.W.; Singh, N. P.; Hart, R. W. **Effects of Chrysotile Co-exposure on BaP Binding in Normal Human Fibroblasts.** *Environmental Health Perspectives* 51: 241-244, September 1983.

Cotreatment with chrysotile (10 μg/mL) and benzo(*a*)pyrene (BaP) produced no observable cytotoxicity after 8 hours of incubation in reseeded high density cell cultures, but did induce cell killing slightly in the confluent cell cultures under serum-free conditions. The effects of cotreatment on uptake, metabolism, and BaP binding to nucleic acids were investigated in normal human fibroblasts. In the presence of serum, there was no observable difference in the amount of water-soluble metabolites recovered between the control and the asbestos-treated cultures. When serum was withheld from the growth medium, a significantly higher percentage of radioactivity was recovered in the aqueous phase of the asbestos-treated medium, indicating that more BaP was metabolized in the treated culture than the control. While it has previously been demonstrated that chrysotile can adsorb BaP easily in the absence of serum, and that the adsorbed BaP was rapidly eluted upon the addition of serum (suggesting that under serum-free conditions chrysotile acts as a vehicle for bringing BaP into cells), this does not appear to be the case when serum is added, which would be more representative of in vivo conditions. (Auth. Abs. Mod.)

84–0376. Chang, W.-C.; Fukuda, S.; Tai, H.-H. **Cigarette Smoking Stimulates Lipoxygenase But Not Cyclooxygenase Pathway in Platelets.** *Biochemical and Biophysical Research Communications* 115(2): 499-505, September 15, 1983.

Cigarette smoke increased 12-lipoxygenase activity significantly without affecting the cyclooxygenase pathway in male rats exposed to freshly generated cigarette smoke once daily for 4 to 8 weeks. Inhalation of smoke was verified by elevated level of carboxyhemoglobin. Arachidonate metabolism through lipoxygenase and cyclooxygenase pathways in platelets was determined. It is concluded that, in view of platelet—leukocyte interactions and potent chemotactic activity in 12(S)-hydroxy-5,8,10,14-eicosatetraenoic acid for aortic smooth muscle cell migration, increased 12-lipoxygenase activity may predispose individuals to atherosclerosis, thromboembolism, and emphysema commonly found in smokers. (Auth. Abs. Mod.)

84–0377. Davis, R. F.; Davis, J. W. **The Effect of Smoking on the Stressed Template Bleeding Time.** *Annals of Clinical Research* 15(3): 131-133, 1983.

Although cigarette smoking is known to affect platelet function, smoking two cigarettes has no effect on the stressed template bleeding time (STBT) of healthy men. The mean initial STBT of 12 nonsmokers (9.0 minutes) was not significantly different from that of nine habitual smokers (9.2 minutes) who were asked to abstain from smoking for 12 hours before determination of the bleeding time. The mean STBT was the same immediately before and after the 21 men smoked two filtered tobacco cigarettes in 20 minutes. Separate analyses showed that the mean STBT of neither the nonsmokers nor the habitual smokers changed significantly after experimental smoking. (Auth. Abs. Mod.)

84–0378. Dossing, M.; Aelum, J. B.; Hansen, S. H.; Lundqvist, G. R.; Andersen, N. T. **Urinary Hippuric Acid and Orthocresol Excretion in Man During Experimental Exposure to Toluene.** *British Journal of Industrial Medicine* 40(4): 470-473, November 1983.

Baseline urinary orthocresol (O-Cr) in smokers was three to four times higher than in nonsmokers among 42 printing trade workers (<10 years exposure to organic solvents including toluene) and among 43 controls matched for smoking habits, age, and living accommodations. To test the efficacy of urinary O-Cr or hippuric acid (HA) in monitoring toluene exposure, matched pairs were randomized to an experimental exposure of either 100 ppm or 0 ppm toluene for 6.5 hours under controlled conditions in an exposure chamber. Baseline urinary O-Cr excretion was higher among smokers than nonsmokers, probably due to the content of O-Cr in cigarettes. The O-Cr excretion in unexposed smokers was, however, 10 times lower than that of the nonsmokers during the end of the experimental exposure to 100 ppm toluene. No difference in HA and O-Cr excretion was found between printing trade workers and controls. The median O-Cr excretion increased 29 times during exposure, whereas the HA excretion increased only five times. Thus, only 3 percent of the O-Cr excretion originated from other sources than toluene, whereas the corresponding value for HA was 19 percent. Standardization of the concentrations of HA and O-Cr in relation to urinary creatinine reduced the relative variation by 29 percent and 56 percent respectively. (Auth. Abs. Mod.)

84–0379. Ebert, R. V.; McNabb, M. E.; McCusker, K. T.; Snow, S. L. **Amount of Nicotine and Carbon Monoxide Inhaled by Smokers of Low-Tar, Low-Nicotine Cigarettes.** *Journal of the American Medical Association* 250(20): 2840-2842, November 25, 1983.

In 76 smokers, correlations between plasma nicotine and alveolar carbon monoxide (CO) levels of the individual smoker and the nicotine and CO yields of the cigarette were very poor. In 24 smokers of low-nicotine, low-tar cigarettes, mean alveolar CO levels did not differ from those of smokers of regular cigarettes. Mean plasma nicotine levels were lower in smokers of low-nicotine cigarettes, but a wide overlap of individual values occurred. The implication that an individual shifting to ultra-low-tar cigarettes reduces risks of cardiopulmonary disease is unwarranted. (Auth. Abs.)

84–0380. Edwards, J. A.; Warburton, D. M. **Smoking, Nicotine and Electrocortical Activity.** *Pharmacology and Therapeutics* 19(2): 147-164, 1983.

The neuropharmacological effects of nicotine and the biological motives for smoking are reviewed. The efficacy of smoking as a means of delivering nicotine to the nervous system is detailed, and nicotine's subsequent neurophysiological activity in both the peripheral and central nervous system is explored. Electrocortical responses to smoking are discussed with respect to the sources of EEG and event-related potential (ERP) activity. Methodological issues covered included sampling, data analysis, definition of controls, physical characteristics of the cigarette, smoking topography, and the relevance of task requirements. Implications of EEG, ERP, and contingent negative variation studies are analyzed in detail.

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84–0381. Elkeles, R. S.; Khan, S. R.; Chowdhury, V.; Swallow, M. B. **Effects of Smoking on Oral Fat Tolerance and High Density Lipoprotein Cholesterol.** *Clinical Science* 65(6): 669-672, December 1983.

Findings are compatible with the hypothesis that smoking interferes with the lipolysis of triglyceride-rich lipoproteins and the conversion of high density lipoprotein $(HDL)_3$ into HDL_2. Changes in serum triglyceride and HDL cholesterol after a fatty meal were studied in seven smokers and seven nonsmokers (mean age 20 years, all less than 100 percent of ideal body weight). While there were no significant differences in initial values between smokers and nonsmokers, among smokers (1) mean serum triglyceride between 0 and 9 hours was greater $(p=0.04)$, and (2) there was a tendency for serum triglyceride to rise higher and remain elevated for a longer period. Data showed a rise in HDL_2 cholesterol in nonsmokers, but not in smokers; this difference was significant at 7 hours $(p=0.02)$. There were no significant differences in HDL_3 cholesterol changes between the groups at 7 hours; however, the HDL_2/HDL_3 cholesterol ratio rose in nonsmokers, but not in smokers, and this difference was significant $(p=0.02)$.

84–0382. Fuxe, K.; Andersson, K.; Eneroth, P.; Siegel, R. A.; Agnati, L. F. **Immobilization Stress-Induced Changes in Discrete Hypothalamic Catecholamine Levels and Turnover, Their Modulation by Nicotine and Relationship to Neuroendocrine Function.** *Acta Physiologica Scandinavica* 117(3): 421-426, March 1983.

Nicotine administration did not affect the serum levels of any of the hormones evaluated in male Sprague Dawley rats. Nicotine counteracted, to a minor degree, the immobilization stress-induced reduction in noradrenaline (NA) levels, and also the stress-induced secretion of ACTH, but not of prolactin, suggesting the involvement of noradrenergic mechanisms, possibly in the paraventricular nucleus in the nicotine modulation of stress-induced increases of ACTH secretion. The nicotine-induced reduction of NA levels in subependymal layer was blocked by stress as well as the tendency for nicotine-induced increases of dopamine turnover in the medial and lateral palisade zones of the median eminence, indicating opposing influences of immobilization stress and nicotine on at least some hypothalamic catecholamine (CA) systems. Immobilization stress (1 hour) induced discrete reductions in NA levels in the posterior periventricular hypothalamic region and in the paraventricular hypothalamic nucleus, and a decrease in dopamine turnover in the medial palisade zone (MPZ) of the median eminence, but failed to induce regional increases of hypothalamic NA turnover. Stress also stimulated the secretion of ACTH, corticosterone and prolactin, while vasopressin, LH, and FSH serum levels were unaffected. The stress-induced reduction of dopamine turnover in MPZ may mediate the stress-induced increase of prolactin secretion. Nicotine (0.3 mg/kg s.c., 1 hour) did not by itself significantly influence CA turnover in the various CA nerve terminal systems analyzed in the hypothalamus, but reduced NA levels in the subependymal layer of the median eminence. (Auth. Abs.)

84–0383. Hartwell, T. D.; Handy, R. W.; Harris, B. S.; Williams, S. R.; Gehlbach, S. H. **Heavy Metal Exposure in Populations Living Around Zinc and Copper Smelters.** *Archives of Environmental Health* 38(5): 284-294, September-October 1983.

In general, smokers had higher metal levels than nonsmokers in hair and blood, and in many cases the differences were significant. Arsenic, cadmium, and lead levels were determined simultaneously in environmental media and human tissues in two zinc smelter and two copper smelter communities. Environmental media sampled included air, soil, household dust, and tap water; human samples included hair, blood, and urine. Between 200 and 300 residents in each community, from various age groups (1-5, 6-18, 20-40, and >60 years) were sampled in 1978 and 1979 and completed questionnaires. Samples for all media were selected under a probability sampling framework at various distances from the smelters. Results indicate that increased environmental levels and body burdens are found closest to the smelters. Of the three tissues sampled, hair was the most useful in determining relationships between environmental metal levels, distance, and body burden. Furthermore, while there was evidence that all ages had hair metal levels related to environmental levels and distance from the smelter, these relationships were much more pronounced for the 1- to 5-year-old age group; this group also had the highest tissue metal levels. Higher hair metal levels were also found for males; children who ate paint, dirt, or clay; and for individuals who spent more time out-of-doors. (Auth. Abs. Mod.)

84–0384. Hassler, E.; Lind, B.; Piscator, M. **Cadmium in Blood and Urine Related to Present and Past Exposure. A Study of Workers in an Alkaline Battery Factory.** *British Journal of Industrial Medicine* 40(4): 420-425, November 1983.

Exposure to airborne cadmium was identical for smokers and nonsmokers employed in an alkaline battery factory but average cadmium concentrations in the blood and urine of smokers were approximately twice as high as those in nonsmokers. Blood and urinary cadmium concentrations together with cadmium in air concentrations from the breathing zone of 18 male workers (9 smokers, 9 nonsmokers) were determined at regular intervals for 11 consecutive weeks. Smokers and nonsmokers did not differ in age or years of employment. Cadmium in air concentrations varied, but no definite trend was observed. The concentrations of cadmium in the blood and urine were found to be stable. For the whole group, urinary cadmium was significantly correlated with years of employment, but no correlation was found between blood cadmium concentrations and exposure. For nonsmokers, the correlation between cadmium in blood and years of employment was statistically significant $(p<0.001)$, indicating that blood concentrations of cadmium reflect body burden in nonsmokers at current low exposure levels. (Auth. Abs. Mod.)

84–0385. Heckman, C. A.; Crankshaw, O. S.; Lehman, G. L. **A Physical Factor Implicated in Morphogenesis of Tobacco Smoke-Induced Pulmonary Lesions.** *Micron* 14(1): 87-88, 1983.

The ultrastructural features of smoke-induced lesions at the bronchiolar-alveolar junction in F344 rats were examined using electron microscopy. Fibrotic thickenings were occasionally found in the septa of alveoli, particularly where macrophages had accumulated in the adventitia of vessels underlying the bronchioles. The macrophages showed numerous irregular, osmiophilic inclusions, and spicule-like, electron-lucent inclusions in the cytoplasm. In some of the lesions, the mucociliary epithelium extended into the respiratory bronchioles and fibrotic alveoli. Both the basal and undifferentiated cells of the epithelium sometimes contained inclusions similar in appearance to those in the macrophages and of a size comparable to carcinogenic fibers. However, analysis of the composition of these inclusions indicated a cellular, rather than an endogenous, origin.

84–0386. Hoffmann, D.; Brunnemann, K. D. **Endogenous Formation of *N*-Nitrosoproline in Cigarette Smokers.** *Cancer Research* 43(11): 5570-5574, November 1983.

Findings suggest that the endogenous *N*-nitrosation of proline, which occurs as a result of cigarette smoke inhalation, may also apply to other *N*-nitrosatable amines including nicotine and thus lead to in vivo formation of carcinogenic *N*-nitrosamines. The potential of inhaled cigarette smoke for endogenous *N*-nitrosation of amines in smokers was assessed by measuring urinary excretion of *N*-nitrosoproline (NPRO) by subjects receiving a controlled diet supplemented by ascorbic acid and proline in measured doses. The urine was analyzed for NPRO, creatinine, and cotinine (the major metabolite of nicotine). The mean 24-hr NPRO excretion for 13 nonsmokers in group 1 was 3.6 μg, while in 13 smokers in group 1 it was 5.9 μg/24 hr, significantly higher than that of the nonsmokers ($p<0.05$). Urinary NPRO in 14 nonsmokers of group 2 was significantly lower than that of the 14 smoking volunteers ($p<0.05$). Data for group 3 indicated that those smokers who had shown elevated NPRO excretion in group 2 had reduced urinary levels of NPRO as a consequence of ascorbic acid intake. Differences in NPRO excretion by smokers and nonsmokers on a controlled diet with ascorbic acid but without proline supplements (group 4) were also significant. (Auth. Abs. Mod.)

84–0387. Holme, J. A.; Dybing, E. **Increased Cytochrome P-450 Independent Drug Metabolism and Mutagen Activation in Rat Liver by Octachlorostyrene.** *Acta Pharmacologica et Toxicologica* 53(4): 325-332, October 1983.

Liver S9 fractions from rats pretreated with a single dose of octachlorostyrene (OCS) showed little effect on benzo(*a*)pyrene (BaP) mutagenicity in the *Salmonella* test. Single intraperitoneal injections of 200 mg/kg OCS increased the activities of flavin-containing monooxygenase, epoxide hydrolase, and glutathione S-transferase in the livers of male Wistar rats. Uridinediphosphate-glucuronyl transferase activities, measured with aglycones increased by methylcholanthrene or phenobarbital treatment, were both slightly increased by OCS treatment. A liver 9,000 X g supernatant fraction from OCS pretreated rats increased the bacterial mutagenicity of 2-acetylaminofluorene and 2-aminofluorene compared with controls, while insignificant or only minor effects were seen on N-hydroxy 2-acetylaminofluorene and BaP mutagenicity. The effect of OCS on mutagen activation was similar to that seen after phenobarbital treatment. The use of monolayers of hepatocytes instead of 9,000 X g subfractions did not reveal any qualitative differences in mutagen activation. (Auth. Abs. Mod.)

84–0388. Hunter, E.; Sloneker, S. D.; Kostenbauder, H. B. **Influence of Chronic Cigarette Smoke Exposure on the Uptake and Efflux of Imipramine in the Isolated Perfused Rat Lung.** *Drug Metabolism and Disposition* 11(4): 361-365, July-August 1983.

Lungs of rats exposed to cigarette smoke exhibited a high pulmonary affinity for imipramine. Using an isolated perfused lung preparation, the lung was perfused for 220 sec with medium containing 2.5 X 10^{-7} M imipramine, followed by a 28-min drug-free perfusion. There was no significant difference ($p<0.05$) between either the rate or the amount of imipramine accumulated in the smoke-exposed and nonexposed animals. During the drug-free perfusion, the previously accumulated imipramine was released from two distinct pools. Calculation of the total amount of effluxable imipramine indicated that in the nonexposed animals, approximately 30 percent of the amount taken up did not efflux at a measurable rate but formed a "noneffluxable" pool. In the smoke-exposed animals, however, all of the accumulated imipramine appeared to be effluxable. The data demonstrate that there may be components of cigarette smoke that are sequestered by lung tissue at the binding sites associated with the noneffluxable pool of imipramine. The presence in cigarette smoke of components possessing such high pulmonary affinity may be a factor in smoke-mediated lung damage. (Auth. Abs.)

84–0389. Iwao, S.; Tsuchiya, K.; Sugita, M. **Variation of Cadmium Accumulation Among Japanese.** *Archives of Environmental Health* 38(3): 156-162, May-June 1983.

Although there was a lack of precise information on smoking habits, cadmium in liver and zinc in liver and renal cortex of males smoking eight or more cigarettes/day were statistically higher than in nonsmokers and those who smoked less than seven cigarettes/day. Human liver, renal cortex, and heart muscle from 394 autopsied Japanese who had lived in eight districts of Japan were sampled. Subjects included 200 males between 0 to 89 years of age and 194 females between 0 and 94 years of age. Cadmium, copper, and zinc in both liver and renal cortex, as well as cadmium in heart muscle were compared by district, sex, age, and smoking habits. Statistical results revealed that the difference of each heavy metal distribution by eight districts is significant and that cadmium and zinc levels in liver and renal cortex sampled from north-central Japan were the highest. This probably results from the greater intake of dietary cadmium and zinc via rice, since residents consume home-grown rice, high in cadmium. Cadmium in renal cortex was not significantly different in the two smoking groups. Further studies should be performed to determine the health significance of an increased intake of cadmium by populations from the viewpoint of global biogeochemistry. (Auth. Abs.)

84–0390. Jaffe, R. L.; Nicholson, W. J.; Garro, A. J. **Urinary Mutagen Levels in Smokers.** *Cancer Letters* 20(1): 37-42, August 1983.

While there was a general correlation between urinary mutagen levels and cigarette consumption, mutagen excretion levels (measured over an 8-week period in 13 smokers and 5 nonsmoking controls) could not be predicted on the basis of either quantity smoked or the tar content of the cigarettes. Among smokers, week-to-week variation of individual urinary mutagen levels was fairly stable. Variability in mutagen levels expressed as the standard error of the mean value was 24 percent or less for 7 of the 13 smokers, 40 percent or less for 11 of the smokers, and in the range of 50 to 60 percent for 2 smokers. Results indicate that genetic or environmental factors, in addition to daily cigarette tar intake, may influence urinary mutagen level magnitude. Controls had no detectable urinary mutagenic activity.

84–0391. Jawaid, M.; Lind, B.; Elinder, C. G. **Determination of Cadmium in Urine by Extraction and Flameless Atomic-Absorption Spectrophotometry. Comparison of Urine From Smokers and Non-Smokers of Different Sex and Age.** *Talanta* 30(7): 509-513, July 1983.

Among 207 randomly selected Swedish subjects, average cadmium urine concentrations in the 42 male and 38 female smokers were twice as high as in nonsmokers of the same age group. The new extraction method described, which determines cadmium in urine by flameless atomic-absorption spectrophotometry after extraction, is much less prone to errors caused by loss of the metal in different steps or by contamination from the reagents. Blood cadmium levels among smokers were about 10 times those found in nonsmokers, reflecting the degree of recent exposure (inhalation). Age was found to have a significant influence on excretion of cadmium. In persons aged 50 to 65, excretion was about twice that found in those under age 35, and thus appears to give a more reliable measure of the body burden than blood cadmium measurements. The median urinary excretion of cadmium for smokers aged 50 to 64 was 0.7 μg/l for males and 0.75 μg/l for females, compared with 0.25 and 0.4 μg/l, respectively, in nonsmokers. Among those aged 19 to 34, levels were 0.35 and 0.3 μg/l for male and female smokers, compared with 0.2 μg/l for both male and female nonsmokers.

84–0392. Kandaswami, C.; O'Brien, P. J. **Effect of Chrysotile Asbestos and Silica on the Microsomal Metabolism of Benzo(*a*)pyrene.** *Environmental Health Perspectives* 51: 311-314, September 1983.

Examination of benzo(*a*)pyrene (BaP) metabolites generated by 3-methylcholanthrene-pretreated rat liver microsomes, in the presence of chrysotile fibers, revealed a reduction in the overall metabolism of the hydrocarbon. Thus, chrysotile appeared to modify the activities of aryl hydrocarbon hydroxylase and epoxide hydrolase. Leaching chrysotile in deionized water for 24 hours elicited a similar response. Silica, in contrast to chrysotile, did not decrease the microsomal metabolism of BaP. Chrysotile, as well as water-leached chrysotile, considerably diminished the microsomal production of water-soluble BaP metabolites. Precoating the fibers with heparin or bovine serum albumin partially abolished this inhibition. The liver microsomal metabolism of BaP in rats subjected to intraperitoneal administration of chrysotile was 58 percent of that in untreated rats. (Auth. Abs. Mod.)

84–0393. Ksir, C. **Taste and Nicotine as Determinants of Voluntary Tobacco Use by Hamsters.** *Pharmacology Biochemistry and Behavior* 19(4): 605-608, 1983.

Syrian hamsters consumed a commercial chewing tobacco in daily amounts equivalent to 2.6 percent of their body weights, even though food and water were always available. Daily intake increased gradually to this level over a period of 4 months. Moistened, unflavored tobacco was consumed in much smaller amounts than the commercial tobacco, which contains flavoring agents and sugars. Addition of sucrose to the unflavored tobacco produced a concentration-dependent increase in daily consumption. Addition of nicotine to the drinking water did not affect daily fluid intake or food consumption, but produced a selective, dose-related decrease in consumption of the commercial tobacco. These results indicate interactive roles for taste and nicotine in controlling the daily, voluntary, high-level use of tobacco by hamsters. (Auth. Abs.)

84–0394. Lloyd, B.; Lloyd, R. S., Clayton, B. E. **Effect of Smoking, Alcohol, and Other Factors on the Selenium Status of a Healthy Population.** *Journal of Epidemiology and Community Health* 37(3): 213-217, September 1983.

Analysis of selenium (Se) status in 391 subjects (266 nonsmokers, 105 smokers, 20 cigar and pipe smokers) in the south of England showed significant effects from smoking, alcohol consumption, age, and use of oral contraceptives. Mean concentrations of glutathione peroxidase (GSH-Px) activity and Se in whole blood, plasma, and erythrocytes were lower in male smokers than nonsmokers. The difference was highly significant in men over age 30. The only significant differences in women were seen between the smokers who were daily drinkers (106.2±11.49 μg/l) and the nondrinking nonsmokers (115.9±16.61 μg/l). Oral contraceptive use resulted in significantly higher Se levels, but had no effect on GSH-Px activity.

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Mean Se in whole blood and erythrocytes was lower in subjects over age 55. Daily alcohol consumption was associated with lower Se in men, but alcohol alone had no appreciable effect on Se status in women.

84–0395. Marks, M. J.; Burch, J. B.; Collins, A. C. **Effects of Chronic Nicotine Infusion on Tolerance Development and Nicotinic Receptors.** *Journal of Pharmacology and Experimental Therapeutics* 226(3): 817-825, September 1983.

Although continuous nicotine infusion (0.2, 1.0, and 5.0 mg/kg/hr) had no effect on baseline rotarod performance or body temperature, the heart rates of mice treated with 1.0 or 5.0 mg/kg/hr of nicotine were significantly lower than those of controls. Significant tolerance to the acute effects of nicotine was noted for both body temperature and rotarod performance. DL-(^{3}H)nicotine binding was measured in seven brain regions. Nicotine infusion resulted in significant increases in binding in cortex, midbrain, hindbrain, hippocampus, and hypothalamus. An increase in α-(^{125}I)bungarotoxin binding was seen in midbrain and hippocampus. No change in L-(^{3}H)quinuclidinyl benzilate binding occurred. Changes resulted from increases in maximum binding site values, whereas K_D values were unaffected. Correlations between nicotine response and binding were sought, and a highly significant correlation between hypothermia and hypothalamic DL-(^{3}H)nicotine binding was obtained. Data indicate that chronic treatment with nicotine results in tolerance to the drug and is accompanied by an increase in brain nicotinic receptors. (Auth. Abs. Mod.)

84–0396. Marshall, M. V.; Noyola, A. J.; Rogers, W. R. **Analysis of Urinary Mutagens Produced by Cigarette-Smoking Baboons.** *Mutation Research* 118(4): 241-256, September 1983.

Urine concentrates from 17 cigarette-smoking baboons and 12 sham puffers were analyzed for mutagenic activity in *S. typhimurium* tester strain TA1538. Both the proportion of animals exhibiting measurable mutagenic activity in urine and the mean level of mutagenic activity present were significantly greater in cigarette-smoking baboons ($p<0.05$). Mutagenic activity in the urine of male and female cigarette-smoking baboons was not significantly different. Age and smoking history did not correlate with mutagenic activity of the urine concentrate from individual animals, but mean blood carboxyhemoglobin did. Fractionation of the urine concentrates on silicic acid separated the concentrate into fractions that were more active in TA100, and others that were more active in TA1538. Further fractionation was accomplished by high pressure liquid chromatography. (Auth. Abs.)

84–0397. Matintalo, M.; Uotila, P. **The Effect of Low-, Medium-, and High-Tar Cigarette Smoke on the Fate of Arachidonic Acid in Isolated Hamster Lungs.** *Acta Pharmacologica et Toxicologica* 53(4): 280-287, October 1983.

Low-, medium-, and high-tar cigarettes affect the fate and metabolism of arachidonic acid in isolated hamster lungs, and effects of medium- and high-tar cigarettes are clearer than those of low-tar cigarettes when the cigarettes are burned with a constant puff volume and rate. Cigarette smoke ventilation increased the amount of ^{14}C-arachidonic acid in the triacylglycerols of perfused hamster lungs but usually had no effect on the amount of radioactivity in diacylglycerols or in different phospholipids. Isolated hamster lungs were ventilated with smoke from low-, medium-, and high-tar cigarettes when 45 nmol of ^{14}C-arachidonic acid was infused into the pulmonary circulation. Most of the infused radioactivity was found in different phospholipid and neutral lipid fractions of the perfused lungs, and a smaller amount was found in the nonrecirculating perfusion effluent, mainly as metabolites. The increased amount of radiolabel in triacylglycerols was significantly greater in the lungs ventilated with smoke from medium- or high-tar cigarettes than in those ventilated with low-tar cigarette smoke. Cigarette smoke ventilation increased the amount of unmetabolized arachidonate in the perfusion effluent and decreased the amount of many metabolites. (Auth. Abs. Mod.)

84–0398. Mikhailidis, D. P.; Barradas, M. A.; Jeremy, J. Y.; Dandona, P. **Cigarette Smoking Inhibits Prostacyclin Formation. (Letter).** *Lancet* 2(8350): 627-628, September 10, 1983.

Another possible mechanism for the reduced ability of chronic smokers to produce vascular prostacyclin (PGI_2) reported earlier (*Lancet* 1(8336): 1248-1250, June 4, 1983) is described. A rise in serum nonesterified fatty acids (NEFA), known to accelerate the rate of decay of PGI_2 in albumin solution and in plasma in vitro, may have several effects related to smoking. It is suggested that an increase in NEFA concentration may contribute to platelet activation, noting that chronic smokers may produce a greater elevation in serum NEFA concentrations than nonsmokers. Chronic smokers may produce less PGI_2 than nonsmokers due to endothelial cell damage (which may be cumulative), also correlated with NEFA concentrations. See also 83-1568

84–0399. Monti-Bloch, L.; Stensaas, L. J.; Eyzaguirre, C. **Carotid Body Grafts Induce Chemosensitivity in Muscle Nerve Fibers of the Cat.** *Brain Research* 270(1): 77-92, June 27, 1983.

Topical application of nicotine (1 to 5 μg) elicited a response in grafted carotid body chemo-mechanosensory fibers, with a discharge frequency of $+32.7\pm3.1$ to $+45.6\pm8.0$ Hz. The ability of carotid body parenchymal cells to change the physiological properties of nerve fibers was tested in eight cats by transplanting the carotid body to the tenuissimus muscle and reinnervating the organ with the muscle nerve. Ninety-five to 174 days after the transplant, electrophysiological recordings were obtained from the reinnervating tenuissimus nerves. Forty percent of the regenerating nerve fibers having spontaneous activity responded to muscle stretch, hypoxia, sodium cyanide, acetylcholine, nicotine, and dopamine, apparently because of dual innervation of glomus cells and muscle spindles. Five of six transplants, examined by electron microscopy, contained viable glomus cells innervated by axon terminals. Neither the normal tenuissimus nerve nor the same nerve regenerating into adipose tissue grafts or to one nonviable carotid body transplant showed chemosensory properties, although these grafts contained numerous, small myelinated axons indicative of extensive nerve regeneration. It is concluded that carotid body cells are capable of inducing chemosensory properties in axons that, normally, subserve a mechanosensory function. (Auth. Abs. Mod.)

84–0400. Mossman, B. T.; Eastman, A.; Landesman, J. M.; Bresnick, E. **Effects of Crocidolite and Chrysotile Asbestos on Cellular Uptake and Metabolism of Benzo(*a*)pyrene in Hamster Tracheal Epithelial Cells.** *Environmental Health Perspectives* 51: 331-335, September 1983.

A rapid transfer of benzo(*a*)pyrene (BaP) to hamster tracheal epithelial cells occurred after addition of ^{3}H-BaP-coated crocidolite and chrysotile fibers to cultures. When BaP was adsorbed to both types of fibers before their addition to cultures, 70 percent of the total BaP introduced entered the cell within 1 hr; 50 percent remained intracellular after 8 hr. In contrast, if identical amounts of BaP were added directly to the medium, an initial influx of 20 percent was observed and cells retained only 5 percent of the initial amount at 8 hr. Little or no alteration in BaP metabolism was observed under these circumstances. Minimal single-strand breakage of DNA was observed after administration of BaP, but not after chrysotile or crocidolite. Moreover, BaP-coated asbestos did not cause DNA breakage in excess of the amounts induced by BaP alone. Results suggest that asbestos not only facilitates the transfer of hydrocarbons into cells, but also increases retention by the "target" cell of the respiratory tract. These processes might be intrinsic mechanisms of asbestos-induced cocarcinogenesis. (Auth. Abs. Mod.)

84–0401. Nakagawa, A.; Chiba, K.; Ishizaki, T.; Nakamura, K. **Influence of Cigarette Smoke on Antipyrine Metabolite Formation in Rats.** *Research Communications in Chemical Pathology and Pharmacology* 41(3): 473-491, September 1983.

Results suggest that the metabolic pathways of antipyrine (AP), a multimetabolized drug, would be affected differently with different experimental inducers, and that smoke and tar act somewhat differently on the hepatic monooxygenase system, resulting in the different profiles of AP metabolites in rat urine. The effects of cigarette smoke on AP disposition in blood and its metabolites in urine were examined in rats before and after pretreatment with tar, 3-methylcholanthrene (3-MC), and phenobarbital (PB). In vitro hepatic monooxygenase activities were also examined in smoke-exposed and tar-pretreated rats and compared to saline-pretreated or control rats. Both the smoke-exposed and tar-pretreated rats showed a significant ($p<0.05$) increase in AP clearance compared with controls. The increases in AP clearance of PB and 3-MC groups were more remarkable ($p<0.01$), being more than three- and eightfold greater than the control group, respectively. The urinary amount of 4-hydroxyantipyrine (4OHA) was significantly ($p<0.05$) greater, and that of norantipyrine (NORA) and AP less ($p<0.05$) in the smoke-exposed rats than in the controls. This trend was not observed in the tar-pretreated rats. In the 3-MC-treated rats, a significant ($p<0.05$) increase in the urinary amount of 4OHA was found, while that of 3HMA was significantly ($p<0.01$) diminished without a change in AP or NORA in the urine. In the PB-pretreated rats, the trend was similar to the 3-MC group except that the amount of unchanged AP was significantly ($p<0.05$) decreased. 7-Ethoxycoumarin O-deethylase was significantly ($p<0.01$) increased in the smoke-exposed rats but not in the tar-treated rats. There was no significant increase in the content of cytochrome P-450 or aminopyrine N-demethylase in the two groups examined. (Auth. Abs. Mod.)

84–0402. Neri, M.; Carugno, N. **Study of the Action of Smoke on Alcohol Dehydrogenase, Lactic Dehydrogenase and Malate Dehydrogenase Enzymes.** *Beitraege zur Tabakforschung International* 12(2): 73-79, June 1983.

The effects of whole smoke and the vapor phase of smoke on yeast alcohol dehydrogenase (YADH), lactic dehydrogenase (LDH), and malate dehydrogenase (MDH) were examined, and the moderating influences of filters and thiol groups tested. Whole smoke inhibited YADH proportionately with number of cigarettes smoked: activity was reduced to 30 percent of original levels with whole smoke from five cigarettes. Addition of cysteine raised activity levels to 45 percent of baseline. Silica or carbon filters showed some protective effects, but cellulose or cellulose acetate filters did not. MDH displayed inhibition by whole smoke, but less than YADH; levels were 40 percent with 5 cigarettes and 20 percent from 10 cigarettes. Vapor phase had little influence, and LDH showed only a negligible inhibition. Data indicate that smoke is highly reactive with the sulfhydrylic groups of the enzymes examined. The minimum quantity of thiol (cysteine) required to obtain maximum protection was estimated at 15 mg/five cigarettes under the experimental conditions.

84–0403. Perricone, R.; De Carolis, C.; De Sanctis, G.; Fontana, L. **Complement Activation by Cigarette Smoke Condensate and Tobacco Infusion.** *Archives of Environmental Health* 38(3): 176-179, May-June 1983.

A significant decrease in measurable total hemolytic complement, classical pathway activity, and alternative pathway activity was observed in all test samples of normal human serum incubated with cigarette smoke condensate (CSC) or tobacco infusion (TI). The capacity of TI and CSC to activate the complement pathway was investigated in vitro by hemolytic and immunological assays. Complement activity was studied using samples incubated at 37°C with different dilutions of TI and CSC. In all mixtures containing TI or CSC at a concentration of 0.1:1, total hemolytic complement showed an average progressive decrease of 90 percent and 50 percent, respectively, after 60 min of incubation. This decrease was associated with a decrease of both classical and alternative pathway activities. In addition, cleavage fragments of C3, C4, and factor B were identified with immunochemical analyses. Results suggest that TI and CSC are activators of complement either through the classical or alternative pathway. (Auth. Abs. Mod.)

84–0404. Peyre, S.; Di Napoli, A.; Pelissero, A.; Sategna-Guidetti, C. **Diagnostic Usefulness of Serum Group I Pepsinogen Determination.** *Gastroenterologie Clinique et Biologique* 7(10): 793-798, 1983.

Serum pepsinogen I (PG-I) levels were significantly higher in smokers among both duodenal ulcer patients (59 smokers, 22 nonsmokers, smoking status of 68 unknown) and controls (92 smokers, 92 nonsmokers). In controls, levels were significantly higher in smokers compared with nonsmokers (69±25 and 56±25 ng/ml, respectively), with no significant difference between males and females. Among males, PG-I levels were higher among smokers than nonsmokers (71±25 and 57±24 ng/ml, respectively), and, although female smokers had higher PG-I values than female nonsmokers (66±26 and 55±27 ng/ml, respectively), the difference was not significant. Smoking did not affect the PG-I fasting level. Among duodenal ulcer patients PG-I was significantly higher in males than in females. A statistically higher PG-I level was also found in duodenal ulcer males compared with control males, while no statistical difference was detected between females with duodenal ulcer and all controls or control females. In the nonsmoking duodenal ulcer group, serum PG-I levels overlapped those of normal controls who smoked. Ranitidine treatment for 1 year did not affect the PG-I. To evaluate clinical usefulness, PG-I was also measured in patients with gastric ulcer, Zollinger-Ellison syndrome, gastrectomy, renal failure, and atrophic gastritis. It is concluded that the most important current clinical application of serum PG-I levels is in screening for the detection of atrophic gastritis, and potential use to detect populations at increased gastric cancer risk.

84–0405. Poole, A.; Brown, R. C.; Fleming, G.T.A. **Study of the Cell-Transforming Ability of Amosite and Crocidolite Asbestos and the Ability to Induce Changes in the Metabolism and Macromolecular Binding of Benzo(*a*)pyrene in C3H10T1/2 Cells.** *Environmental Health Perspectives* 51: 319-324, September 1983.

Results showed that asbestos dusts were capable of augmenting the oncogenic effect of benzo(*a*)pyrene (BaP). This synergistic effect was evident when fibers and chemicals were added to cultures as simple mixtures and when BaP was adsorbed to the surface of fibers. Asbestos dust did not, however, appear to exert its oncogenic-enhancing effect by modifying the metabolism of BaP in C3H10T1/2 cells. Crocidolite and amosite caused no increase in the number of transformed colonies over that seen in cultures from untreated cells. (Auth. Abs. Mod.)

84–0406. Recio, L.; Enoch, H.; Hannan, M. A. **Parameters Affecting the Mutagenic Activity of Cigarette Smokers' Urine.** *Journal of Applied Toxicology* 2(5): 241-246, 1982.

While no nonsmoker's urine showed any mutagenic activity in Ames tests, the mutagenicity of smoker's urine varied from 31 to 162 revertants/plate. The dose—response relationship between number of cigarettes smoked and urine mutagenicity, and persistence of mutagenic activity after smoking cessation were examined. Although a dose—response relationship (urine mutagenicity vs. the number of cigarettes smoked/day) was not observed in the smokers assayed, a linear dose—response relationship was detected in an individual smoker who smoked a varying number of cigarettes over 8 successive days. In another smoker who ceased smoking, mutagens were detected in the urine for 1 week. It is noted that the urine concentrate of three low-tar cigarette smokers showed weak activity (31 to 50 revertants/plate, mean=40/plate), suggesting that mutagenicity is related to tar exposure. (Auth. Abs. Mod.)

84–0407. Roure, H. **Extrait de Tabac de l'Institut Pasteur (11 Ans d'Etude - 3 Etapes). [Tobacco Extract From the Pasteur Institute (11 Year Study in Three Phases).]** In: Molimard, R. (Editor). *1^{ere} Journee de la Dependance Tabagique.* Comptes Rendus, U.E.R. Biomedicale des Sainte-Peres, Paris, December 11, 1982, pp. 103-112, French.

Immune system responses to tobacco were examined over a period of several years, using a denicotinized tobacco extract diluted to 0.001. In the first phase, 10 nonsmoking patients with respiratory troubles and reaginic reactions were treated: a tolerance to tobacco smoke was obtained in some of the patients treated with tobacco extract. In the second phase, heavy smokers exhibiting clinical respiratory symptoms had positive delayed reaction to intradermic tests. In the third phase, auricular therapy was applied to heavy smokers by injecting diluted tobacco extract into the subjects willing to stop smoking. This technique, called mesoisotherapy, appeared to be useful in smoking cessation or prevention. Detoxification by mesoisotherapy was applied to 13 male and 4 female patients with various symptoms of illness or in an early stage of pregnancy. After two to four sessions, 10 patients stopped smoking and 7 reduced their consumption. Very good results were observed in four patients 3 months later, and good results in two patients 1 year later.

84–0408. Sivarajah, K.; Jones, K. G.; Fouts, J. R.; Devereux, T.; Shirley, J. E.; Eling, T. E. **Prostaglandin Synthetase and Cytochrome P-450-Dependent Metabolism of (±)Benzo(*a*)pyrene 7,8-Dihydrodiol by Enriched Populations of Rat Clara Cells and Alveolar Type II Cells.** *Cancer Research* 43: 2632-2636, June 1983.

Enriched fractions of Clara cells and alveolar type II cells from rat lung tissue metabolized (±)-*trans*-7,8-dihydroxy-7,8-dihydrobenzo(*a*)pyrene (BaP-7,8-diol) to BaP diol-epoxide I, a highly carcinogenic compound. The metabolism of BaP-7,8-

diol by prostaglandin synthetase and cytochrome P-450-dependent monooxygenases was examined in the presence of arachidonic acid or NADPH, using control and β-napthoflavone (β-NF)-pretreated rats. Results indicate that (1) BP-7,8-diol was oxidized exclusively to BaP diol-epoxide I in fractions enriched in both cell types and by both enzymes; (2) the oxidation of BaP-7,8-diol differed with the different cell fractions; and (3) in both enzyme systems, the metabolism to BaP diol-epoxide I was inducible by β-NF treatment. It is suggested that pulmonary prostaglandin synthetase may serve as either an additional or an alternative bioactivating enzyme to the cytochrome P-450-dependent monooxygenases for the formation of reactive chemical carcinogens in the lung.

84–0409. Sorsa, M.; Maki-Paakkanen, J.; Vainio, H. **A Chromosome Study Among Worker Groups in the Rubber Industry.** *Scandinavian Journal of Work, Environment and Health* 9(Supplement 2): 43-47, 1983.

Smoking correlated with increased frequencies of sister-chromatid exchanges and chromosome aberrations among both referents (n=35) and rubber workers (n=55) representing six job categories in two rubber factories. Two cytogenetic endpoints, structural chromosome aberrations and sister-chromatid exchanges, were analyzed in the blood lymphocytes of several worker groups and referents to evaluate possible work-related exposures to chromosome-damaging agents. Two job categories, mixers and weighers of chemicals, showed statistically significant increases in sister-chromatid exchange values. When chromosome aberrations were analyzed, only the group of nonsmoking weighers showed significantly higher values than the reference group. (Auth. Abs. Mod.)

84–0410. Stankowska-Chomicz, A. **Wplyw Palenia Tytoniu na Metabolizm Lekow. [The Effect of Smoking Tobacco on the Metabolism of Drugs.]** *Polski Tygodnik Lekarski* 37(23): 689-691, July 19, 1982, Polish.

Cigarette smoke, a pharmacologically active substance, has a two-phase effect on several metabolic processes that take place in the liver. The initial phase involves a reduction in the rate of biotransformation, while the later phase involves the induction of microsomal proteins of the liver, and probably stepped-up synthesis of metabolizing enzymes. Studies carried out on humans and animals indicate that the primary effect of smoking cigarettes is an accelerated transformation of drugs, caused by the induction of microsomal enzymes in the liver. Smoking's effect on the metabolism of several drugs, and the impact of polycyclic aromatic hydrocarbons on liver protein synthesis, blood circulation, phospholipid synthesis, and other hepatic functions, are described.

84–0411. Stephens, R. E.; Joseph, L. B.; Daniel, F. B.; Schenck, K. M.; Newman, H.A.I.; Lipetz, P. D.; Millette, J. R. **Interactions of Chrysotile and Benzopyrene in a Human Cell Culture Systems.** *Environmental Health Perspectives* 51: 257-265, September 1983.

When applied on normal human fibroblasts at concentrations of 100 μg/mL, National Institute of Environmental Health Sciences (NIEHS) short chrysotile asbestos was more cytotoxic than NIEHS intermediate chrysotile (3 percent and 17 percent survival, respectively). Benzo(*a*)pyrene (BaP) and benzo(*e*)pyrene (BeP) concentrations up to and including 10 μM were not cytotoxic. Simultaneous application of NIEHS short chrysotile with BaP or with NIEHS intermediate chrysotile resulted in increased survival over that of intermediate chrysotile alone (25 percent and 17 percent survival, respectively). There were low levels of BaP bound to DNA in the presence of NIEHS short chrysotile or NIEHS intermediate chrysotile. Measurable levels of BaP-DNA adducts were formed both in the absence and in the presence of each size of NIEHS chrysotile. However, there was no strong indication of a perturbation of the level of DNA-BaP binding following simultaneous administration of increasing levels of asbestos in addition to 1 μM hydrocarbon. The asbestos had no demonstrable influence on the level of BaP metabolism during the 24-hour period following simultaneous exposure of asbestos and hydrocarbons. In addition, the BaP—deoxynucleoside adducts formed both in the presence of 1 mg/mL short chrysotile and in its absence, as determined by cochromatography on high pressure liquid chromatography with standards formed by the reaction of the (+)antibenzo(*a*)pyrenediol epoxide at the exocyclic amino group of deoxyriboguanosine (N^2). (Auth. Abs. Mod.)

84–0412. Stults, B. M.; Hashisaki, P. A. **Influenza Vaccination and Theophylline Pharmacokinetics in Patients With Chronic Obstructive Lung Disease.** *Western Journal of Medicine* 139(5): 651-654, November 1983.

Cigarette smoking did not significantly affect theophylline clearance after standard influenza vaccination in patients with stable chronic obstructive lung disease. Total theophylline clearance rate was measured before and 24 hours after influenza vaccination in seven men (five smokers and two ex-smokers, mean age 64 years). In four subjects, total theophylline clearance rate was also measured 48 hours after vaccine administration. There was no significant change in the clearance rate after either time interval. These results do not support recent recommendations to monitor serum theophylline concentrations or reduce theophylline dosage during the 48-hour period following influenza vaccination. Nevertheless, pending further studies, patients maintained on a regimen of theophylline preparations should be followed clinically for theophylline toxicity for the first several days after receiving influenza vaccine. (Auth. Abs. Mod.)

84–0413. Tirlapur, V. G.; Gicheru, K.; Charalambous, B. M.; Evans, P. J.; Mir, M. A. **Packed Cell Volume, Haemoglobin, and Oxygen Saturation Changes in Healthy Smokers and Non-Smokers.** *Thorax* 38(10): 785-787, October 1983.

The relationship between cigarette smoking, packed cell volume, hemoglobin concentration, and arterial oxygen saturation (SaO_2) was investigated in 114 nonsmokers, 66 light smokers (1 to 20 cigarettes/day), and 50 heavy smokers (>20 cigarettes/day), aged 20 to 75 years. Packed cell volume was significantly greater in female heavy smokers over 40 years of age and in all female smokers over 60 years than in nonsmokers. Hemoglobin concentrations were higher in 40- to 59-year-old female heavy smokers ($p<0.05$) and in male and female light ($p<0.05$) and heavy smokers ($p<0.001$) over 60 years of age than in nonsmoking contemporaries. Compared with nonsmokers, SaO_2 was lower in 20- to 39-year-old male heavy smokers ($p<0.02$) and female ($p<0.05$) light smokers and in 40- to 59-year-old male light and heavy smokers ($p<0.001$) and female light smokers ($p<0.02$). It was also lower in female light ($p<0.05$) and heavy ($p<0.02$) smokers over age 60; and higher in male light smokers over 60 ($p<0.001$). Changes in SaO_2 were seen at a younger age than changes in hemoglobin concentration and packed cell volume. Data show that smokers (compared with nonsmokers) generally have mild erythrocytosis similar to that seen in relative polycythemia, particularly in older people. (Auth. Abs. Mod.)

84–0414. Tripathi, O. N. **Arecoline Induced Nicotinic and Muscarinic Stimulation of the Superior Cervical Ganglion of Cat.** *Biomedica Biochimica Acta* 42(2/3): 275-282, 1983.

Arecoline (10-500 µg) stimulated the nicotinic receptors in the superior cervical ganglion (scg) of cat following intra-arterial injection into the ganglion. The ganglion stimulating effect of arecoline was weaker than that of dimethylphenylpiperizinium (DMPP) but stronger than that of pilocarpine. Similarly, the time course of response to arecoline was slower than that of DMPP but faster than that of pilocarpine. The gangliomimetic effects of arecoline, nicotine, and DMPP are prevented by hexamethonium, tetraethylammonium, and high doses of nicotine and arecoline. Eserine potentiated the response to arecoline, but not that to DMPP and nicotine. Response to submaximal preganglionic stimulation and KCl were potentiated by arecoline and pilocarpine, but not by DMPP and nicotine. The responses to arecoline and pilocarpine were also prevented by atropine, which did not alter the responses to DMPP and nicotine. It is suggested that arecoline stimulated the muscarinic as well as nicotinic receptors in the scg of cat, and that acetylcholine is likely to be partially involved in the gangliomimetic effects of arecoline. (Auth. Abs. Mod.)

84–0415. Vadlamani, N. L.; Pontani, R. B.; Misra, A. L. **Effect of Chronic Nicotine Pre-Treatment on Phencyclidine (PCP) Disposition in the Rat.** *Archives Internationales de Pharmacodynamie et de Therapie* 265(1): 4-12, September 1983.

Nicotine pretreatment did not affect blood or tissue distribution of phencyclidine (PCP), but did significantly potentiate PCP-induced locomotor activity. Rats treated either with saline or 1 mg/kg nicotine twice daily for 11 days were administered 5 mg/kg (^{3}H)PCP. Nicotine-treated rats showed lower plasma concentrations of PCP metabolites 30 minutes after PCP injection, but no other significant differences in PCP distribution were observed. Relative locomotor activity, however, was significantly elevated in the nicotine-treated rats between 0.5 and 2.0 hours after PCP administration. It is suggested the enhanced physical activity involves an additive interaction at the central nervous system level.

84–0416. Wiersma, D. A.; Roth, R. A. **Total Body Clearance of Circulating Benzo(*a*)pyrene in Conscious Rats: Effect of Pretreatment With 3-Methylcholanthrene and the Role of Liver and Lung.** *Journal of Pharmacology and Experimental Therapeutics* 226(3): 661-667, September 1983.

The metabolic clearance of benzo(*a*)pyrene (BaP) was studied in conscious control and 3-methylcholanthrene (3-MC)-pretreated rats, and the roles of liver and lung in total body BaP clearance were estimated. During a 5-hour period, blood BaP concentration decreased rapidly in a biphasic manner. 3-MC pretreatment did not significantly affect the rate constants of BaP elimination, but the apparent volume of distribution increased, resulting in an increase in total body clearance from 15±1 to 48±6 ml/min. Five hours after injection, BaP concentrations in most organs were lower in 3-MC-pretreated rats; however, lung BaP concentration was greater. In the 3-MC-pretreated rats, first-pass extraction of BaP by lung and liver was determined from comparison of areas under arterial blood BaP concentration vs. time curves after i.a., i.v., and intrahepatic portal venous administration. The liver first-pass extraction was 20 percent compared with 33 percent by the lung. Organ BaP concentrations were correlated with the route of BaP administration. Results suggst that (1) 3-MC pretreatment increases the apparent volume of BaP distribution in vivo; (2) the liver and lung of 3-MC-pretreatment rats are about equal in their ability to extract circulating BaP in vivo; and (3) 3-MC pretreatment enhances total body BaP clearance by increasing extrahepatic BaP elimination. (Auth. Abs.)

84–0417. Wright, J. H.; Whitaker, S. B.; Welch, C. B.; Teller, D. N. **Hepatic Enzyme Induction Patterns and Phenothiazine Side Effects.** *Clinical Pharmacology and Therapeutics* 34(4): 533-538, October 1983.

Smokers had higher levels of D-glucaric acid (D-ga) excretion among both psychotic patients and control subjects treated for 12 days with phenothiazines. Of the 32 patients, 22 were smokers while only 5 of the 15 controls were smokers. Mean D-ga levels reached 11.7±8.2 $\mu g/l/m^2$ in smokers, compared with 7.5±3.7 $\mu g/l/m^2$ ($p<0.05$) in nonsmokers. About half the psychiatric patients had a treatment-related rise in D-ga excretion compatible with enzyme induction. These patients had fewer and less severe neurologic side effects than those who did not have a significant rise in urinary D-ga levels. Smoking was unrelated to enzyme induction.

See also, **84–0422, 84–0563, 84–0568, 84–0598**

MORTALITY AND MORBIDITY

84-0418. Conti, S.; Farchi, G.; Menotti, A. **Coronary Risk Factors and Excess Mortality From All Causes and Specific Causes.** *International Journal of Epidemiology* 12(3): 301-307, September 1983.

Followup data from two Italian rural cohorts of the Seven Countries Study associated smoking with 17 percent of total mortality (42 percent of cancer deaths and 32 percent of coronary heart disease deaths), suggesting that smoking has a multipotential role. The association of stroke deaths with smoking was not significant. The analysis considered the rural cohorts of Crevalcore in northern Italy and Montegiorgio in central Italy (n = 1,712 males, aged 40 to 59 years at entry in 1960). The 15-year followup for life status and causes of death (n = 378) was complete for all men: coronary heart disease (n = 78 deaths), stroke (n = 42), cancer (n = 116), other (n = 142). The excess risks of death attributable to blood pressure, smoking, and serum cholesterol, and the estimated risk of death, are reported. Analysis indicated that blood pressure was associated with a substantial amount of total mortality, while serum cholesterol seemed to be a specific risk factor for coronary mortality. (Auth. Abs. Mod.)

84-0419. Denman, D. W., III.; Schlesselman, J. J. **Interval Estimation of the Attributable Risk for Multiple Exposure Levels in Case—Control Studies.** *Biometrics* 39(1): 185-192, March 1983.

In case—control studies, exposure to a risk factor (such as smoking) often occurs at several levels, so the attributable risk at each level is important. In an example of such exposure, the estimated attributable risk of smoking for myocardial infarction (MI) in women aged 35 to 39, and the relationship with oral contraceptive use, was determined. Tabulated data show estimated smoking rates, estimated odds ratio, and estimated attributable risk, which was 0.259 in women smoking 1 to 24 cigarettes/day, and 0.562 in those smoking more than 25 cigarettes/day. Thus, an estimated 26 percent of MI cases were attributable to smoking less than 25, and 56 percent attributable to smoking more than 25 cigarettes/day. For the reference group of nonsmokers, the attributable risk was 0.179, so 18 percent of cases were not attributable to smoking. Estimation for the 2 X 2 table (case—control status vs. dichotomous exposure) and the 2 X *k* table (case—control status vs. exposure at several levels) is reviewed. A method for finding confidence intervals for attributable risk in the 2 X *k* table is proposed, and its application to estimates adjusted across strata is indicated. The results of a Monte Carlo study of the procedure demonstrate that the nominal and actual coverage probabilities agree satisfactorily for practical applications. (Auth. Abs. Mod.)

84-0420. Hopkin, J. M.; Gorecka, D. **Tough Cells and Old Age.** *Lancet* 2(8360): 1170-1173, November 19, 1983.

Variations in individual responses to the cytotoxicity of cigarette smoke may be an important determinant of emphysema risk among heavy smokers. To resolve whether differing responses were related to disease development, subjects were divided into two groups: (1) 55-year-old smokers (n = 10) with severe bronchitis and emphysema; and (2) controls (n = 10) matched for age, sex, and smoking habits, with no evidence of respiratory disease. Controls were more resistant to cytotoxic effects of cigarette smoke than the disease group at all levels of testing. Results suggest that differences between the groups might be primary. Furthermore, eight subjects with various inflammatory disorders of the lung and other systems (including pneumonia and bronchiectasis) were found to have highly resistant cellular responses, refuting the possibility that the results for the disease group were simply secondary to the presence of inflammatory change. Further testing of 100 healthy individuals, aged 20 to 94 years, indicated a progressive loss of the spectrum of cellular response to cigarette smoke, corresponding with advancing age. Individual responses to acetaldehyde, tested at the beginning of the study, were identical. Of subjects aged 65 years or over, 70 percent were females; 60 percent were current or ex-smokers; and all had been "social drinkers," with approximately 20 percent consuming alcohol regularly. The study included persons with mild degenerative central nervous system or cardiovascular disease. While physiological, cellular, and biochemical functions generally decline in the elderly, cells of elderly subjects tested performed better than their younger counterparts. It is suggested that (1) individuals with increased cellular resistance also have increased cellular longevity and slower cell cyclings, arriving later at the finite number of divisions where organic dysfunction and death occur; and (2) emphysema may be a natural aging process, accelerated by cigarette smoke due to its tendency to kill lung cells and to hasten cell turnover toward its finite number of divisions.

84-0421. Kuller, L. H.; Radford, E. P. **Epidemiological Bases for the Current Ambient Carbon Monoxide Standards.** *Environmental Health Perspectives* 52: 131-139, October 1983.

A review of the basis for current ambient carbon monoxide (CO) standards emphasizes that cigarette smoking is the major source of CO, and separation of smoking-related CO from that of other sources is difficult. The health impacts of CO vary with atmospheric concentration, exposure duration, respiratory ventilation, blood volume, barometric pressure, diffusibility from the lung, rate of endogenous CO production, and the ratio of the affinity of blood for CO or O_2. Evidence of an association between CO levels and acute heart attack, coronary atherosclerosis, elevated hospital admissions, general cardiovascular complaints, electrocardiographic changes, and fetal effects from a variety of community and experimental studies are reviewed. Sources of CO discussed include automobile exhaust emissions, occupational sources, urbanicity, home heating sources, and seasonal factors. It is concluded that the evidence does not suggest the CO standard is too high, although a reduction in cigarette smoking should be a high public health priority.

84–0422. Peters, G. **La Toxicologie de la Fumee de Tabac et les Maladies des Fumeurs. [The Toxicology of Tobacco Smoke and Smokers' Diseases.]** *Therapeutische Umschau/Revue Therapeutique* 40(2): 148-156, February 1983, French

The toxic constituents of tobacco and cigarette smoke (nicotine, carbon monoxide, hydrocyanic acid, condensates, nitrosamines, and residues) are discussed. Each is examined in terms of its quantity in smoke, pharmacological effects and the organs affected, and toxicity. Cigarette smoking leads to a considerable increase in diseases such as lung cancer, and probably arteriosclerosis of the limbs and coronary arteries. Smoking also results in a less marked increase in other diseases such as cancer of the bladder and digestive tract, gastritis, and duodenal ulcer, although it is debatable whether cigarette use is correlated with disorders of vision and hearing. Some of these effects can be ascribed to nicotine or to the constituents of tar and cigarette smoke, although nicotine is probably not the most toxic constituent of smoke. It is noted that while the incidence of many cancers is higher among smokers than nonsmokers, no positive proof of a causal relationship exists for cancers other than those of the lung. (Auth. Abs. Mod.)

84–0423. Rickert, W. S. **"Less Hazardous" Cigarettes: Fact or Fiction?** *New York State Journal of Medicine* 83(13): 1269-1272, December 1983.

While smokers of low-yield cigarettes may be at reduced risk of death from lung cancer, myocardial infarction risk is not lower and overall health risks are reduced far less than anticipated. Low-yield cigarettes are often as hazardous as higher yield brands due to the tendency, ascribed to nicotine dependency, to block filter vents (a practice admitted by half of low-tar cigarette smokers) and to increase puff volume and/or depth of inhalation. Furthermore, air dilution devices reduce mainstream yields to the smoking machine, but not sidestream yields to the environment. In addition, human smoking behavior is so variable that it is doubtful whether a single set of conditions for machine smoking can provide reasonable values for smoke constituents such as hydrogen cyanide (HCN). The range of constituent levels for a single cigarette brand is greater than the range recorded for all 115 cigarette brands tested. Nevertheless, response of health conscious consumers to low-tar brands has been extraordinary, with the low-tar ($\leq$ 15 mg) segment in the United States tripling from 17 percent in 1976 to 59 percent in 1982. A similar pattern has been observed in Canada. Changes in cigarette yields from 1969 to 1982 are described, and health effects of CO, carboxyhemoglobin levels, nicotine, aldehydes, and HCN are discussed.

84–0424. Rubin, G. L.; Ory, H. W.; Layde, P. M. **The Mortality Risk of Voluntary Surgical Contraception.** *AVS Biomedical Bulletin* 3(2): 1-4, December 1982.

Cumulative mortality risk for women starting oral contraceptive use at age 15 is estimated at 12 for nonsmokers and 356 for smokers at age 35, a 30-fold difference. In contrast, voluntary surgical contraception (VSC) mortality risks are lower than those of a single pregnancy, prolonged oral contraceptive use, or smoking a pack of cigarettes/day. Mortality risk for female VSC is approximately 4/100,000 procedures in developed countries and 16.2/100,000 procedures in less developed countries. Mortality risk for vasectomy is between 0.1 and 19.0/100,000 procedures. It is noted that the small risk of VSC is limited primarily to the first 6 weeks after the procedure.

84–0425. Schievelbein, H. **Rauchen und Praventivmedizin. [Smoking and Preventive Medicine.]** *Offentliche Gesundheitswesen* 44(11): 712-717, November 1982, German.

Cessation of smoking is the best preventive measure available against many illnesses; however, since many smokers appear to be unable to quit, the development of less dangerous cigarettes is deemed necessary. Nicotine is considered a relatively harmless substance in the doses consumed by smokers, and lowering the tar content while maintaining nicotine levels is recommended. It is noted that the tar and nicotine yields of cigarettes have been cut in half over the past 10 years, and that the market share of filtered cigarettes has increased steadily. Smoking of lower yield cigarettes is evaluated as positive: some studies have shown that smokers of light cigarettes have lower mortality rates and lower risks of cancer compared with smokers of medium- and high-yield cigarettes, although still higher than those of nonsmokers. Graphs showing mortality rates, relative risks of lung and laryngeal cancer, and peripheral vascular disease are provided.

See also, **84–0436**

NEOPLASTIC DISEASES

84–0426. Ames, B. N. **Dietary Carcinogens and Anticarcinogens. Oxygen Radicals and Degenerative Diseases.** *Science* 221(4617): 1256-1264, September 23, 1983.

Colon, breast, and lung cancers, which are almost entirely due to smoking, account for about half of all cancer mortality in the United States. Humans consume a great variety of natural mutagens and carcinogens, as well as antimutagens and anticarcinogens. Several grams of burnt and browned material may be ingested from the diet, and smokers additionally inhale 500 mg daily from two packs of cigarettes at 0.20 mg tar/cigarette. Both smokers and those who have consumed a meal of fried pork or bacon have more easily detectable levels of mutagens in urine. Sixteen specific examples of naturally occurring mutagens and carcinogens in food are discussed, including catechols that are also present in cigarette smoke. Anticarcinogens are also considered, and cooked food as a source of burnt material is explored. The role of oxygen radicals in degenerative diseases and the aging process, and other additional sources of risk, such as cosmetics and environmental pollution, are reviewed.

84–0427. Bergman, H.; Axelson, O. **Passive Smoking and Indoor Radon Daughter Concentrations. (Letter).** *Lancet* 2(8362): 1308-1309, December 3, 1983.

In response to a recent article (*Lancet* 2(8350): 595-597, September 10, 1983) that noted an association between maternal smoking and lung cancer in current smokers, and between passive smoking and lung cancer, data are presented regarding the importance of radon decay products in indoor air. Results showed that the concentration of radon daughters (a risk factor for lung cancer among miners) nearly doubled in an unoccupied room when 12 cigarettes were burned by machine. When air is stirred by people moving, concentrations may well increase. It is suggested that children of mothers who smoke may receive early exposure to high levels of radon daughters at home, and that smoking in later life promotes the development of lung cancer. See also 84-0434

84–0428. Bhatia, P. L.; Bhide, S. V. **Risk Factors in Oesophageal Cancer.** *Indian Journal of Cancer* 20(1A): 43-48, March 1983.

Analysis of 26 cases of esophageal cancer in Manipur showed smoking, betel chewing, and possibly a diet high in smoked, fermented, and coarse foods as risk factors. Heavy bidi and tobacco smoking (>20 bidis/day for >25 years) was found in 83.3 percent of patients; 29.8 percent chewed tobacco, and 7.8 percent had been betel chewers for 30 to 40 years. Local tobacco contained 1.8 percent nicotine by dry weight, but no nitrosamines. Alcohol consumption (brewed from rice) was noted in 33.3 percent of patients. Rice is the staple diet, and large quantities of chilies (family Solanaceae), and other fried, smoked, fermented, and spicy foods are also consumed.

84–0429. Binnie, W. H.; Rankin, K. V.; Mackenzie, I. C. **Etiology of Oral Squamous Cell Carcinoma.** *Journal of Oral Pathology* 12(1): 11-29, February 1983.

Tobacco use is clearly an etiologic factor in oral cancer, the risk varying with form of consumption. Pipe, cigar, and cigarette smoking, reverse smoking, chewing (loose leaf, pan, and betel quids), and snuff-dipping all represent different risk ratios. It is noted that cigarette use presents a relatively small risk compared with chewing and pipe smoking, and replacement of cigarette smoking with "less harmful" forms of tobacco consumption creates an increased risk of oral cancer. Also, a synergistic relationship between tobacco and alcohol is evident. In addition, interrelationships among iron deficiency, chronic candidiasis, herpes simplex virus, syphilis, and oral neglect are reviewed as factors in oral cancer.

84–0430. Bohm, G. M. **Air Pollution and Lung Cancer.** *Cancer Detection and Prevention* 5(4): 371-374, 1982.

Knowledge of the correlations between cigarette consumption and lung cancer, tobacco carcinogenesis, and other data have failed to reduce smoking, whereas a single nuclear accident created global awareness of that hazard. The risk of long-term and low-level exposure to carcinogens is perceived as acceptable by the public, which seeks immediate benefits and ignores remote hazards. Experimental work has provided understanding of (1) physics of particle deposition in the lungs (inertial impact, sedimentation, and Brownian movement); (2) carcinogenesis, through studies of bay region theory (polycyclic aromatic hydrocarbons) and surface charges (asbestos); (3) cocarcinogenesis of atmospheric particulates once accepted as harmless (iron and benzo(*a*)pyrene); and (4) in vitro screening methods for malignant potential and pathogenesis, such as bacterial mutation tests, organ cultures, and sister-chromatid exchange systems. Cigarette smoke remains the most serious problem among the many carcinogenic air pollutants. More enlightenment on smoking is needed. (Auth. Abs. Mod.)

84–0431. Bruzzi, P.; Margarino, G.; Tonetti, R.; Bonelli, L.; Catturich, A.; Scala, M. **Uno Studio Caso-Controllo su Fattori di Rischio dei Tumori del Cavo Orale. Prospettive ed Indicazioni per la Diagnostica Precoce. [A Case-Controlled Study on the Risk Factors of Oral Cavity Tumours: Prospects and Indications for Early Diagnosis.]** *Minerva Medica* 74(1/2): 19-24, January 14, 1983, Italian.

The literature on carcinomas of the oral cavity shows general agreement that alcohol and smoking are risk factors. Only a few authors blame poor hygiene. A retrospective survey conducted in Genoa evaluated the relative importance of these risk factors, by means of a case—control study linking a specific risk factor to a specific condition. A similar survey was conducted on a control group. For the 98 histologically confirmed cases hospitalized in 1979-1980, smoking and alcohol, often together, were found to be the most significant risk factors in the oncological pathology of the oral cavity. (Auth. Abs.)

84–0432. Buhler, D. R.; Unlu, F.; Thakker, D. R.; Slaga, T. J.; Conney, A. H.; Wood, A. W.; Chang, R. L.; Levin, W.; Jerina, D. M. **Effect of a 6-Fluoro Substituent on the Metabolism and Biological Activity of Benzo(*a*)pyrene.** *Cancer Research* 43(4): 1541-1549, April 1983.

Comparison of the tumor-initiating activities of benzo(*a*)pyrene (BaP) and 6-fluorobenzo(*a*)pyrene (FBP) on the skin of female Sencar mice indicates that the fluorinated hydrocarbon is less active in the initiation of tumors. The metabolites of the fluorinated 7,8-dihydrodiol do not display strong mutagenic activity toward Chinese hamster V79 cells. Altered conformation of the fluorinated 7,8-dihydrodiol and the resultant 7,8-diol-9,10-epoxide diastereomer in which the benzylic 7-hydroxyl group and the epoxide oxygen are *trans* provides a plausible explanation for the weak tumorigenicity of FBP on mouse skin, since all benzo-ring dihydrodiols and diol-epoxides in which the hydroxyl groups prefer the pseudodiaxial conformation are weak or inactive as carcinogens. In formulating the bay-region theory, the perifluorine substituents such as at position 6 in BaP have a marked inhibitory effect on the tumorigenicity of the hydrocarbon. (Auth. Abs. Mod.)

84–0433. Coggon, D.; Acheson, E. D. **Trends in Lung Cancer Mortality.** *Thorax* 38(10): 721-723, October 1983.

No other known cause of lung cancer explains the recent trends in mortality as well as smoking. Britain has had an epidemic of lung cancer during this century, yet differences between the sexes in the timing of the epidemic suggest that environmental factors are not the underlying cause. As the effects of the carcinogen are manifest below the age of 40, it must begin to act by early adult life. Differences between urban and rural lung cancer rates may be due to increased occupational risks. However, smoking is more common in city dwellers, and those in the city are likely to have smoked longer. Although still increasing in the elderly, death rates are falling in the youngest age groups, probably due to reductions in tar content of cigarettes and the use of filter tips. When all ages are considered, the death rate is stabilizing in men but still rising among women, because smoking became popular among women later than among men. The death rate will continue to rise for 10 years if current trends are maintained.

84–0434. Correa, P.; Pickle, L. W.; Fontham, E.; Lin, Y.; Haenszel, W. **Passive Smoking and Lung Cancer.** *Lancet* 2(8350): 595-597. September 10, 1983.

Questions about the smoking habits of parents and spouses were asked in a case—control study involving 1,338 lung cancer patients and 1,393 comparison subjects in Louisiana. Nonsmokers married to heavy smokers had an increased risk of lung cancer, as did subjects who had mothers who smoked. There was no association between lung cancer risk and paternal smoking. The association with maternal smoking was found only in smokers and persisted after controlling for variables indicative of active smoking. It is not clear whether the results reflect a biological effect associated with maternal smoking or the inability to control adequately for confounding factors related to active smoking. (Auth. Abs. Mod.)

84–0435. Davis, J. M. **Self-Inflicted Disease.** *Pennsylvania Medicine* 86(11): 28-30, November 1983.

Thirty percent of all U.S. cancer deaths are related to smoking, and current estimates place the costs attributable to smoking in Pennsylvania at 17 billion dollars annually. Approximately one in three (2.4 million) Pennsylvanian adults are smokers. Cigarette use is declining among men but not in women. Since current smoking trends among women began, oral cancer incidence has escalated, bladder cancer incidence has increased by 50 percent, pancreatic cancer rates have approached those of men, and lung cancer is expected to surpass breast cancer as the most common neoplastic disease in women. While 95 percent of ex-smokers quit on their own, it is emphasized that a doctor's advice, support, and encouragement are important. Physicians need to consistently deliver strong antismoking messages. Strategies and sources of effective materials are suggested.

84–0436. Greenberg, M.; Barrows, D.; Clark, P.; Grohs, S.; Kaplan, S.; Newton, N. **White Female Respiratory Cancer Mortality. A Geographical Anomaly.** *Lung* 161(4): 235-243, June 1983.

It is suggested that a pronounced shift of highest white female respiratory cancer mortality rates, compared with other cancer types, from Northeastern and East North Central to Southern and Western regions be analyzed with respect to (1) substantial, increasing differences in female cigarette smoking both within and between metropolitan areas; and (2) geographical diffusion of tobacco smoking from Northeastern and North Central to other regions and suburbs/rural areas of metropolitan regions (particularly Cincinnati, Houston, Los Angeles/Long Beach, Indianapolis, San Francisco/Oakland, and Washington, D.C./Baltimore, defined as group II cities). It is stated that the key to understanding the geographical anomaly presented by white female respiratory cancer rates exists in the central cities. Central cities of seven regions in which there were higher suburban than central city increases in female respiratory cancer mortality rates (Chicago, Cleveland, Denver, Detroit, Milwaukee, New York City/Jersey City/Newark, and Philadelphia, defined as group I) experienced relatively small increases in respiratory cancer rates. The average age-adjusted rate in the group I cities was 26 percent higher than the national rate in 1950-1954; two decades later, after increases averaging 103 percent (from 6.57 to 13.37/100,000), the average age-adjusted rate in these central cities was the same as the national rate. In contrast, group II cities experienced an average increase in the age-adjusted rate of 237 percent (from 5.53 to 18.65/100,000). The average rate in the group II cities climbed from 6 percent higher than the national rate and 16 percent lower than the group I cities' average rate in 1950-1954, to 39 percent higher than both the national rate and the group I cities' average rate by the early 1970s.

84–0437. Heraud, G.; Billaux, M. S. **Alimentation et Cancer. [Nutrition and Cancer.]** *Gazette Medicale de France* 89(23, Supplement): 1-13, June 18, 1982, French.

Tobacco and alcohol consumption are cited as responsible for 100,000 deaths/year in France. The role of tobacco, food, and alcohol consumption in the etiology of cancers of the larynx, esophagus, stomach, pancreas, bladder, and lung, is reviewed. The relative risk of laryngeal and esophageal cancer from simultaneous use of cigarettes and alcohol was calculated, and thiocyanates are discussed as possible etiologic agents in gastric cancer. French research on pancreatic cancer is summarized, including smoking as a factor. Tryptophan metabolites and benzopyrenes are cited as carcinogens responsible for bladder cancer. It is noted that combined use of tobacco and opium has resulted in a risk factor of 19 for bladder cancer in Iran.

84–0438. Hickey, R. J.; Allen, I. E. **Surgeons General's Reports on Smoking and Cancer: Uses and Misuses of Statistics and of Science. (Letter).** *Public Health Reports* 98(5): 410-411, September-October 1983.

The use and misuse of statistics in an overview of the Surgeon General's report (*Public Health Reports* 97(4): 318-324, July-August 1982) are discussed, noting that while statistical methods can reject an hypothesis, statistics alone can never establish that an hypothesis is certainly true. The authors are criticized for not considering or evaluating competing hypotheses (such as the possible genetic relationship between the tendency to smoke and the tendency to develop lung cancer). Such hypotheses cannot be rejected or validated by opinion, judgment, or popularity. It is also pointed out that smoking may be symptomatic of a physiological deficiency that nicotine tends to alleviate. See also 83-0073.

84–0439. Hillerdal, G.; Karlen, E.; Aberg, T. **Tobacco Consumption and Asbestos Exposure in Patients with Lung Cancer: A Three Year Prospective Study.** *British Journal of Industrial Medicine* 40(4): 380-383, November 1983.

A total of 435 patients with bronchial carcinoma of World Health Organization types I to IV were given a questionnaire to determine occupation, smoking habits, and exposure to asbestos. Chest X-rays were screened for the presence of pleural plaques. Of the men, 96.2 percent were current or ex-smokers, as were 71 percent of the women. In those who had never smoked, 70 percent had an adenocarcinoma. In smokers, the risk of getting other types was greater, but the risk of getting an adenocarcinoma was also considerably increased compared with the normal population. Of the men, 35.8 percent were occupationally exposed to asbestos and 15.6 percent were carriers of radiological plaques, a frequency five to six times greater than expected. Practically all asbestos-exposed patients with lung tumors were smokers or ex-smokers and their total tobacco consumption was as high as that of nonexposed patients. The average latency from first exposure to asbestos to diagnosis of the lung tumor was 37 years. Even if strict regulations are warranted for the use of asbestos, this cannot prevent future asbestos cancers resulting from exposure that has already occurred. Elimination of smoking seems the only way to reduce such tumors. (Auth. Abs.)

84–0440. Jardin, A.; Vallancien, G. **Tumeurs de Vessie et Tabac. [Tumors of the Bladder and Tobacco.]** *Gazette Medicale de France* 90(7): 545-547, February 18, 1983, French.

The prevalence of bladder tumors in male smokers is statistically obvious. This relationship is directly dependent on the amount of tobacco consumed, as in the case of carcinoma of the lung. Nevertheless, analysis of the association between bladder cancer and tobacco consumption remains difficult. The carcinogenic substances present in tobacco and its breakdown products are undoubtedly multiple and poorly known, and research is complicated by the existence of other risk factors in addition to tobacco. In view of the gravity of bladder tumors, for which no satisfactory treatment presently exists, prevention by widespread education of the public and doctors concerning the bladder cancer risk from tobacco is necessary. (Auth. Abs. Mod.)

84–0441. Lee, P. N. **Lung Cancer and the "Safer" Cigarette.** *Practitioner* 227(1383): 1459, 1461, September 1983.

The overall reduction in cigarette tar delivery may be a major factor in the decline in lung cancer rates, but it is possible that past exposure to high-yield cigarettes masks many of the benefits of tar reduction. However, reducing the tar dose by 50 percent does not result in the same risk reduction as halving the number of cigarettes smoked/day, and risk reduction from lower tar content will occur only if consumption does not increase. It is estimated that one high-yield nonfiltered cigarette from the past equals three or four modern low-yield cigarettes, providing a much lower lifetime tar exposure. Recent data show that the average reduction in mortality for low-tar smokers is 25 to 30 percent compared with smokers of high-tar or nonfiltered brands. It is noted that since risk is also related to duration of smoking, an increased duration will overwhelm any benefit from smoking low-tar cigarettes.

84-0442. Mayo, P.; Saha, S. P.; Mayo, W. **Primary Lung Cancer in Patients Under 40: A Collective Review of 304 Patients.** *Journal of the Kentucky Medical Association* 81(10): 759-762, October 1983.

The majority of the 33 lung cancer patients in one of the groups studied were smokers (26 of the 28 whose smoking history was known), averaging 1.5 packs/day for 10 years. While lung cancer in patients under the age of 40 is rare, it must seriously be considered in every instance of radiologic lung lesion of indefinite etiology. The stage of the disease in these patients (all under 40 years of age) at the time of diagnosis was far advanced, the resection rate low, and survival rate was poor (4.4 percent 5-year survival in the collected series of 304 such patients). The incidence of adenocarcinoma and small cell undifferentiated carcinoma was high. Prompt diagnosis and resection of the tumor offers the best prognosis, while prevention could be best realized by major changes in smoking habits. (Auth. Abs. Mod.)

84-0443. McGuirt, W. F. **Snuff Dipper's Carcinoma.** *Archives of Otolaryngology* 109(11): 757-760, November 1983.

A trend among younger age groups to switch from cigarettes to snuff may not decrease tobacco-related cancer incidence, but merely change the site from lung and larynx carcinoma to carcinoma of the oral cavity. Of 290 North Carolina patients with primary oral cavity or oropharyngeal tumors, 57 used snuff and no other known oral carcinogenic agents. They were primarily white women, over 60 years of age, who had "dipped" snuff for over 40 years. Carcinomas appeared predominantly in the buccal (47.5 percent) and alveolar (31.5 percent) regions, coincident to the location of the "quid." Many that clinically looked like verrucous carcinomas were histologically well-differentiated squamous cell carcinomas, which acted as other squamous cell carcinomas of the oral cavity. Irradiation or resection were equally effective for the same grade of tumor. Fifty-eight percent of patients had recurrence or a contiguous second primary tumor; almost half (47.3 percent) had concurrent leukoplakia, and 13.9 percent had leukoplakia excised previously, all related to the progressive panmucosal insult. (Auth. Abs. Mod.)

84-0444. Mommsen, S.; Aagaard, J.; Sell, A. **A Case-Control Study of Female Bladder Cancer.** *European Journal of Cancer and Clinical Oncology* 19(6): 725-729, June 1983.

Multivariate logistic analysis showed cheroot smoking to be associated with a relative risk (RR) of 3.28 for bladder cancer in women. A case—control study was performed on 47 female bladder cancer patients and 94 female controls matched for age and geographic area. Twenty-five determinants of assumed importance were examined. The bivariate associations revealed a significantly increased RR for bladder cancer associated with use of tobacco, cheroot smoking, and saccharin consumption, and in some subgroups, such as never smokers, in combination with use of saccharin. RR for cheroot smokers who also used saccharin increased to 20.03. A significant trend for increased bladder cancer risk with increased daily consumption was noted for lifetime cigarette or cheroot consumption. (Auth. Abs. Mod.)

84-0445. Pham, Q. T.; Gaertner, M.; Mur, J. M.; Braun, P.; Gabiano, M.; Sadoul, P. **Incidence of Lung Cancer Among Iron Miners.** *European Journal of Respiratory Diseases* 64(7): 534-540, October 1983.

Miners who died from lung cancer (n=13) were of similar age and had worked for about the same length of time as the 27 who died from other causes between 1975 and 1980, but consumed more cigarettes and had significantly higher venous blood levels of carboxyhemoglobin. A random sample of 1,173 iron miners from Lorraine, France (35 to 55 years of age), were studied by questionnaire, clinical examination, and lung function tests. The incidence of lung cancer was more than three times the number expected in the French male population in the same age groups. All subjects who died from lung cancer were underground miners and, at the first examination, had a higher incidence of bronchitis and more functional defects than both the 27 who died from other causes and the underground miner group as a whole. The excess mortality due to lung cancer is discussed with regard to noxious agents that were measured (radioactivity, which was rather low, total and respirable dusts, nitrogen oxides, carbon monoxide) and with regard to both the work environment and duration of work. (Auth. Abs. Mod.)

84-0446. Pukkala, E.; Teppo, L.; Hakulinen, T.; Rimpela, M. **Occupation and Smoking as Risk Determinants of Lung Cancer.** *International Journal of Epidemiology* 12(3): 290-296, September 1983.

Data on the smoking habits of 1,328 males in different occupational categories in Finland show that variation in smoking prevalence between different occupational groups closely corresponds with that in the standardized incidence ratio (SIR) for lung cancer (R=0.96). Compared with the risk of the total economically active population, the relative risk of those not active (SIR) was 1.69 for men and 0.86 for women. Lower than expected relative risks were encountered among highly educated and white-collar male workers (religious, legal, pedagogical, medical, technical, and administrative); in sales and transport service work; and among farmers. High SIRs were found in mining and quarrying, forestry, woodworking (joiners), construction, painting, and among unskilled workers. Among women, incidence was low and the only significant SIR obtained showed a lower risk than expected in farming. Data on all cases of lung cancer diagnosed in Finland in age groups 35 to 69 years (1971 to 1975) were supplemented by information on occupation from the 1970 census (Central Statistical

Office). The expected numbers of cases were based on the sex, age, and occupation-specific numbers of person-years computed in the Central Statistical Office, and sex- and age-specific incidence rates of lung cancer among the economically active population (as defined January 1, 1971). (Auth. Abs. Mod.)

84–0447. Sanz Ortiz, J.; Farinas Alvarez, M. C.; Gomez Serrano, M.; Mendez Batan, M. A. **Relacion del Habito del Tabaco con los Diferentes Subtipos Histologicos del Carcinoma Broncopulmonar. [Histological Subtypes of Bronchopulmonary Carcinoma Related to Cigarette Smoking.]** *Medicina Clinica* 80(4): 154-156, February 12, 1983, Spanish.

The association between cigarette smoking and histological subtypes was studied in 86 histologically diagnosed Spanish bronchopulmonary carcinoma patients. Overall, 88 percent of the cases were smokers (75 of 76 men and 1 of 10 women). A significant association was found between smoking and epidermoid carcinoma (91 percent smokers), adenocarcinoma (82 percent smokers), and small-cell anaplasia (94 percent smokers). Of 10 cases of large-cell anaplasia, 80 percent smoked (not statistically significant). Significant associations were also found for duration of smoking habit (only 1 patient smoked <10 years, 6 between 10 and 20 years, and 69 >20 years), and for cigarette consumption/day among epidermoid and adenocarcinoma patients. Data showed significant differences among epidermoid and adenocarcinoma cases who smoked >20 cigarettes/day and those smoking <20/day. For the small-cell carcinomas, the differences in the duration and amount of consumption were not significant.

84–0448. Selikoff, I. J.; Frank, A. L. **Keynote Address: Potential of *In Vitro* Tests in Asbestos-Related Problems.** *Environmental Health Perspectives* 51: 1-4, September 1983.

Lung cancer mortality is multiplied in asbestos workers who smoke (20 percent of all deaths in some cohorts), and a similar interaction is a factor in increased esophageal, pharyngeal, buccal cavity, and laryngeal cancer incidence. However, both smoking and nonsmoking asbestos workers are at approximately equal risk for pleural or peritoneal mesothelioma, stomach or colonic cancer, and kidney neoplasms. Lung cancer mortality risk among asbestos workers who quit smoking is reduced within 5 to 10 years to approximately one-half to one-third the risk of continuing smokers, suggesting a need to clarify cellular developments underlying this risk reversal. Further study is needed regarding (1) the nature of the cellular responses of carcinogens (singly and together) that culminate in neoplastic change; and (2) the nature of cellular differences among organs that result in varying response to cigarette smoke and asbestos combined. Additional asbestos-related diseases, neoplastic change, and mortality are discussed.

84–0449. Stevens, M. H.; Gardner, J. W.; Parkin, J. L.; Johnson, L. P. **Head and Neck Cancer Survival and Life-Style Change.** *Archives of Otolaryngology* 109(11): 746-749, November 1983.

Survival among 200 patients with head and neck cancers (oral cavity, pharynx, larynx) was lowest in patients who continued to smoke, while persistent alcohol consumption did not affect survival. A retrospective analysis used patient and family questionnaires and chart review; 69 patients with tumors at head and neck sites known to be unrelated to tobacco and alcohol use served as controls. Alcohol use alone increased the risk of acquiring cancer of the oral cavity, pharynx, or larynx 3.6 times; smoking alone increased the risk 5.8 times. Both factors combined increased the risk 19 times. Those who continued to smoke after diagnosis had a fourfold increase in the recurrence rate over those who did not smoke, and double that of those who stopped smoking. (Auth. Abs. Mod.)

84–0450. Thomas, D. C. **Statistical Methods for Analyzing Effects of Temporal Patterns of Exposure on Cancer Risks.** *Scandinavian Journal of Work, Environment and Health* 9(4): 353-366, August 1983.

In multivariate logistic analysis of the relative risk of lung cancer due to smoking and asbestos exposure, the direct effect of duration of smoking was so strong that when this variable was added to the equation, the contribution of total smoking became nonsignificant. The multistage model, incorporating simple exposure indices, together with additional temporal variables and their interactions, was compared with direct fitting to models of the disease process. Case-referent data on 10,939 males from the Quebec crysotile-asbestos mortality study were analyzed. From the multistage model, average age at exposure, age at risk, and duration of exposure were found to contribute direct or interaction effects. Two models of carcinogenesis were fit. In a two-stage initiation-latency model the median latency period was estimated at 11.0 years overall, but was somewhat longer for asbestos-induced and shorter for smoking-induced tumors. When this difference was allowed for, latency periods seemed to be inversely related to the average intensities of both asbestos and smoking. In the multistage model, the best fit was obtained with asbestos acting at the fourth and smoking at the fifth of six stages. Lifetime risks of lung cancer are projected under the different models. (Auth. Abs. Mod.)

84–0451. Tuyns, A. J. **Oesophageal Cancer in Non-Smoking Drinkers and in Non-Drinking Smokers.** *International Journal of Cancer* 32(4): 443-444, October 15, 1983.

Among nondrinking male smokers, the relative risk for esophageal cancer is over 5, but risk is even higher in nonsmoking male drinkers and increases with rise in daily intake. This effect was also found in females. Alcohol and tobacco are the two major risk factors for esophageal cancer, and among the 743 cases studied (704 males, 39 females) only 11 of the males had never consumed alcohol, and only 38 had never smoked. Among the 39 females, 36 had never smoked, but only 8 had never consumed alcohol. There were no cases reported among nondrinking female smokers. The unusually high sex ratio in the study population from Calvados, France, may reflect the differential distribution of drinking and smoking in males and females; males drink more and smoke much more than females. Data confirm that this is a likely explanation, since the risk values at various levels of consumption are practically identical in both sexes.

84–0452. Van Wyk, C. W. **The Etiology of Oral Cancer.** *Journal of the Dental Association of South Africa* 37(8): 509-512, August 1982.

Review of etiologic factors in oral cancer reveals that nearly every research worker in the field has considered the tobacco habit to be the most important factor in this group of diseases. From available information it is apparent that oral cancer is a multifactorial disease. Most heavily implicated are tobacco, alcohol, sunlight, and nutritional deficiencies. In a state of deficiency brought on by a defective diet, liver damage (cirrhosis), or both, the oral tissues are more susceptible to carcinogenic agents. When tobacco, which contains such agents, is brought into contact with oral tissues, there is an appreciably greater risk of developing cancer. The exception appears to be lip cancer, which is primarily caused by sunlight.

84–0453. Varkey, B. **Asbestos Exposure. An Update on Pleuropulmonary Hazards.** *Postgraduate Medicine* 74(4): 93-99, 102-103, October 1983.

Cigarette smoking increases the risk of bronchogenic carcinoma in persons exposed to asbestos, and some data suggest that the risk is multiplicative. People are still being exposed to thousands of asbestos-containing products that could lead to asbestosis, malignant neoplastic diseases, and nonmalignant pleural disorders. Clinical and roentgenographic manifestations of asbestosis mimic other diffuse fibrotic lung diseases. However, a concomitant finding of pleural plaques strongly suggests the diagnosis. The manifestations of bronchogenic carcinoma related to asbestos exposure are varied and do not differ from those unrelated to asbestos exposure, while malignant mesothelioma signals previous asbestos exposure. Nonmalignant pleural disorders may not cause any symptoms but should alert the physician to other concurrent or potential asbestos-related diseases. To diagnose asbestos-related diseases, physicians should bear in mind the long latent period and should take a careful, comprehensive occupational and environmental history. Cigarette smoking also increases the mortality due to asbestosis. (Auth. Abs. Mod.)

84–0454. Vutuc, C.; Kunze, M. **Tar Yields of Cigarettes and Male Lung Cancer Risk.** *Journal of the National Cancer Institute* 71(3): 435-437, September 1983.

Of 252 male lung cancer patients, 248 were cigarette smokers, significantly more than among 839 controls (n=526, 64 percent). In relation to the relative risks (RR) of never smokers, the adjusted (for age, total years of smoking, and average number of cigarettes smoked/day) lung cancer RR for smokers who had exclusively smoked cigarettes belonging to group II (15 to 24 mg tar/cigarette) was 10.4; for smokers who had exclusively smoked cigarettes belonging to group III (>24 mg tar/cigarette), RR was 25.1. The respective RRs of smokers who had mainly smoked cigarettes belonging to group I (<15 mg tar/cigarette), group II, or group III were 10.9, 20.6, and 36.7. After accounting for differences in daily consumption, the adjusted lung cancer RRs for smokers who had consumed mainly cigarettes belonging to the various groups were: group II (11 to 20 cigarettes/day) compared with group III (11 to 20 cigarettes/day), RR=0.6; group II (>20 cigarettes/day) compared with group III (>20 cigarettes/day), RR=0.8; group II (>20 cigarettes/day) compared with group III (11 to 20 cigarettes/day), RR=1.3; group II (>20 cigarettes/day) compared with group III (< 10 cigarettes/day), RR=7.8; and group II (11 to 20 cigarettes/day) compared with group III (<10 cigarettes/day), RR=2.5. (Auth. Abs.)

84–0455. Vyas, J. J.; Deshpande, R. K.; Sharma, S.; Desai, P. B. **Multiple Primary Cancers in Indian Population: Metachronous and Synchronous Lesions.** *Journal of Surgical Oncology* 23(4): 239-249, August 1983.

The relationship between lesion sites, time intervals, histological variations, and habits such as tobacco and pan chewing were analyzed in patients attending the Tata Memorial Hospital, who (1) had reported with multiple primary neoplasms (n=38), or (2) had single primary lesions and developed second primary lesions at a later time (n=139). Of the 177 subjects, 49 were tobacco/pan chewers, 37 used tobacco and lime quids, and 40 smoked cigarettes and bidis (16 also consumed moderate to large amounts of alcohol). All patients who later developed other primary cancers had continued their tobacco habits after treatment for the original neoplasm. The involvement of physiologically and anatomically related organs in multiple (concurrently or at an interval) primary cancers is observed and explored.

84–0456. Wald, N. J.; Idle, M.; Boreham, J.; Bailey, A. **Inhaling and Lung Cancer: An Anomaly Explained.** *British Medical Journal* 287(6401): 1273-1275, October 29, 1983.

A hitherto unresolved anomaly, that the risk of lung cancer is less among heavy cigarette smokers who say they inhale than among those who say they don't, is explained by results of tests on 2,108 male cigarette smokers. An objective index of inhaling cigarette smoke, based on carboxyhemoglobin concentrations and the carbon monoxide yields of cigarettes, was used to investigate possible systematic differences in the extent of inhaling among light and heavy smokers when classified according to their self-described inhaling habits. Heavy smokers (20 or more cigarettes/day) had a higher average inhaling index than light smokers (fewer than 20 cigarettes/day) both among those who said that they inhaled and among those who said that they did not. There is also indirect evidence that heavy smokers who inhale deeply may to some extent avoid depositing smoke condensate on their main bronchial epithelium. (Auth. Abs. Mod.)

84–0457. Walter, S. D.; Iwane, M.; Flanders, W. D.; Rothman, K. J. **Interaction of Alcohol and Tobacco in Laryngeal Cancer. (Letters).** *American Journal of Epidemiology* 117(5): 639-641, May 1983.

In a reanalysis of earlier data on the association of alcohol consumption and tobacco use with laryngeal cancer (*American Journal of Epidemiology* 115(3): 371-379, March 1982), using a log-linear model rather than categorized frequency data, it was found that a concise description of the findings is possible with either model, although the descriptions differ. However, neither data set is sufficiently extensive to distinguish a preference between the additive and multiplicative statistical analyses. The original authors reply that their goal was to evaluate the extent to which exposure to both alcohol and tobacco represents a greater public health hazard than the sum of the risks of exposure to either factor alone. Since the multiplicative model cannot address the question of public health interaction, it was not considered. See also 82-0964.

84–0458. Weiss, W.; Benarde, M. A. **The Temporal Relation Between Cigarette Smoking and Pancreatic Cancer.** *American Journal of Public Health* 73(12): 1403-1404, December 1983.

Data show that temporal trends in cigarette smoking prevalence rates in the United States from 1920 to 1978 were related to temporal trends in pancreatic cancer mortality rates in both sexes. The differences in cancer rates were rather small in the 1930s, and the curves diverged thereafter to an extent that would be expected if differences in smoking prevalence were a significant etiologic factor. In males, the rise and fall in smoking prevalence were followed by a rise and recent fall in pancreatic cancer rates. In females, the later rise in smoking prevalence has been accompanied by a rise in cancer rate, and a recent slight decline in smoking rates has been associated with a slowing of the rise in the cancer rate. (Auth. Abs. Mod.)

84–0459. Whittemore, A. S.; McMillan, A. **Lung Cancer Mortality Among U.S. Uranium Miners: A Reappraisal.** *Journal of the National Cancer Institute* 71(3): 489-499, September 1983.

Differences between uranium miners' and nonminers' lung cancer mortality rates were substantially higher for smokers than for nonsmokers in a group of 3,362 white miners surveyed in 1974 and 1977, who had been medically examined between 1960 and 1970. The analytic methods represent a miner's annual age-specific lung cancer mortality rate as the (unspecified) rate among nonsmoking men born at the same time and with no mining history, multiplied by the relative risk factor R. This factor depends on the miner's total exposures to radon daughters (in working level months) and to cigarettes (in packs), accumulated from start of exposure until 10 years before his current age. A multiplicative relative risk function specifies that ratios of mortality rates for miners vs. nonminers with similar age and smoking characteristics do not depend on smoking status. The data rejected ($p=.01$) several additive functions for R that specify relative risk as a sum of components due to radiation and to cigarette smoking. Cumulative exposures to both radiation and cigarettes gave better fits to the data than did average annual exposure rates. Age at start of underground mining had no effect on risk, after controlling for age at lung cancer death, year of birth, and cumulative radiation and smoking exposures. (Auth. Abs. Mod.)

84–0460. Winters, T. H.; DiFrenza, J. **Radioactivity and Lung Cancer in Active and Passive Smokers.** *Chest* 84(6): 653-654, December 1983.

Evidence from other studies has shown that radiation is a major factor in lung cancer etiology, and while smoking provides a constant source of intense alpha radiation for the life of a smoker, passive smokers are also exposed to the naturally occurring radioisotope, radon, and its decay products, radon daughters. The passive smoker is also exposed to the same radioelements in tobacco as the smoker (50 to 70 percent of the polonium-210 appears in sidestream smoke). In addition, the exposure of the passive smoker to naturally occurring radon daughters is increased in a smoky environment. Knowledge of the presence of radioactivity in cigarette smoke, in the setting of increased public concern about radiation, presents a more tangible threat than "tar" in motivating people to quit smoking. However, it is noted that scare tactics do not work for all smokers, and the problem will probably be solved only when smoking is considered socially unacceptable.

84–0461. Wynder, E. L.; Goodman, M. T. **Epidemiology of Breast Cancer and Hair Dyes.** *Journal of the National Cancer Institute* 71(3): 481-488, September 1983.

Smoking was positively associated with hair dye use in a case—control study of 399 breast cancer patients (114 smokers, 93 ex-smokers, 192 nonsmokers) and 621 controls. Smoking incidence was higher (74.7 percent) among hospitalized controls than cancer patients (71.9 percent), undoubtedly reflecting the strong association of smoking with a variety of diseases requiring hospitalization. No association was found between hair dye use and breast cancer (odds ratio = 1.02) or any factors affecting dosage, frequency, duration, type, or color of hair dye. Religion was also an important confounder of hair dye use. When stratified by 10-year age groups, there was no appreciable trend in risk status between cases and controls and no dose—response relationship was found, even after controlling for factors affecting hair dye use. A multiple logistic regression analysis supported the absence of this relationship. (Auth. Abs. Mod.)

84–0462. Wynder, E. L.; Goodman, M. T. **Smoking and Lung Cancer: Some Unresolved Issues.** *Epidemiologic Reviews* 5: 177-207, 1983.

The general question of smoking and health continues to be a challenging field for epidemiologists, for those interested in mechanisms of carcinogenesis, cardiovascular disease, and pulmonary emphysema, and particularly for the health educator. This literature review focuses on aspects of lung cancer epidemiology, providing an historical perspective and summation of the current state of knowledge before discussing unresolved questions of current interest. Topics covered include (1) histologic patterns, (2) passive smoking, (3) the low-yield cigarette (and smoking compensation), and (4) nutritional deficiencies or imbalances (role of vitamin A and of β-carotene, and cholesterol). Smoking prevention strategies reviewed are (1) primary prevention, (2) smoking cessation activities, and (3) the role of the low-yield cigarette. Eventually, society will develop a set of priorities regarding health education, and scientists who know about disease risk factors such as smoking should feel bound to apply this knowledge to benefit society. An extensive bibliography is provided.

See also, **84–0485, 84–0492, 84–0623**

NON—NEOPLASTIC RESPIRATORY DISEASES

84–0463. Bisson, G.; Drapeau, G.; Lamoureux, G.; Cantin, A.; Rola-Pleszczynski, M.; Begin, R. **Computer-Based Quantitative Analysis of Gallium-67 Uptake in Normal and Diseased Lungs.** *Chest* 84(5): 513-517, November 1983.

A computer-based method of quantification of gallium-67 (^{67}Ga) lung uptake was used to process data from 36 normal patients (13 smokers), and 11 patients with chronic obstructive pulmonary disease, 28 with biopsy-proven pulmonary sarcoidosis, 33 with asbestosis, and 16 with silicosis. Background radioactivity below the kidneys was subtracted from counts in all areas and given a score of 0; the maximal liver uptake was scored as 16. Radioactivity over six lung fields was scored by the computer between 0 and 16 and averaged to yield a mean ^{67}Ga lung uptake index. This index of ^{67}Ga lung accumulation correlates with histopathologic scores of inflammation in lung tissue and with bronchoalveolar lavage levels of ^{67}Ga radioactivity retrieval. In normal subjects below age 40, the index was significantly higher in smokers than in nonsmokers. Results of the quantitative analysis of ^{67}Ga lung uptake in normal subjects and patients with various pulmonary diseases are reported. The method provides an objective index of inflammatory activity in lung tissues, and is simple, sensitive, and discriminative. (Auth. Abs. Mod.)

84–0464. Bitterman, P. B.; Adelberg, S.; Crystal, R. G. **Mechanisms of Pulmonary Fibrosis. Spontaneous Release of the Alveolar Macrophage-Derived Growth Factor in the Interstitial Lung Disorders.** *Journal of Clinical Investigation* 72(5): 1801-1813, November 1983.

Patients with interstitial pulmonary disorders had alveolar macrophages that spontaneously released a fibroblast growth factor independent of influence by smoking habits. Alveolar macrophages (suspension culture, 4 hr, 37°C) from 65 patients (30 smokers) and 30 controls (13 smokers) were examined for the spontaneous release of fibroblast growth-promoting activity, with human lung fibroblasts as the target. Whereas none of the controls had macrophages spontaneously releasing a growth-promoting activity for fibroblasts, 82 percent of the patients with interstitial lung disease had alveolar macrophages that were spontaneously releasing a growth-promoting activity for fibroblasts. Smoking did not significantly affect growth-promoter release in sarcoidosis patients (smokers 180 ± 44 units, nonsmokers 120 ± 34 units; $p < 0.1$) or in idiopathic pulmonary fibrosis patients (smokers 301 ± 40 units, nonsmokers 291 ± 38 units; $p < 0.3$). (Auth. Abs. Mod.)

84–0465. Burrows, B.; Lebowitz, M. D.; Barbee, R. A.; Knudson, R. J.; Halonen, M. **Interactions of Smoking and Immunologic Factors in Relation to Airways Obstruction.** *Chest* 84(6): 657-661, December 1983.

The relationships of smoking, allergy skin test reactivity, and serum immunoglobulin (IgE) to ventilatory function were analyzed in 1,182 subjects (aged 35 or over) from a general population sample. Impairment of the forced expiratory volume in 1 sec (FEV_1) shows a definite relationship with smoking and total serum IgE. However, the IgE relationship is significant only in smokers with a low FEV_1 accompanied by symptoms, suggesting asthmatic disease or chronic bronchitis. Allergy skin test reactivity to a battery of common aeroallergens shows no overall relationship with FEV_1. However, after accounting for total serum IgE, positive allergy skin tests tend to be associated with high rather than low FEV_1 values. The findings suggest that some type of IgE not specific for aeroallergens but associated with smoking may help explain the "chronic asthmatic bronchitis" syndrome. (Auth. Abs. Mod.)

84–0466. Casterline, C. L. **Interaction of Immunoglobulin E and Cigarette Smoke. Predisposition to Symptomatic Lung Disease?** *Chest* 84(6): 652-653, December 1983.

Immunoglobulin E (IgE) patterns in cigarette smokers include a striking rise in serum IgE in light smokers and a drop in heavy smokers, reversible after cessation of smoking. A recent study (*Chest* 84(6): 657-661, December 1983) investigated correlations between immediate-type hypersensitivity parameters (IgE levels and prick tests) and forced expiratory volume in 1 sec (FEV$_1$). Positive prick tests were associated with elevated FEV$_1$, elevated IgE with low FEV$_1$, and elevated IgE with asthma or chronic bronchitis, indicating that elevated IgE is clinically correlated with development of symptomatic lung disease. These correlations remain unexplained, but may be due to hypersensitivity to an unknown antigen, possibly a component of tobacco or smoke. Further investigation of humoral and cellular immune systems of smokers is needed, particularly among the symptomatic low FEV$_1$, high IgE subpopulation. See also 84-0466

84–0467. Colby, T. V.; Lombard, C. **Histiocytosis X in the Lung.** *Human Pathology* 14(10): 847-856, October 1983.

Although the etiology of pulmonary histiocytosis X remains unknown, data from three recent series suggest a relationship with smoking. Of pulmonary histiocytosis X patients, 80 percent in one recent study were smokers; 80 percent in a separate series were smokers at time of diagnosis (97 percent had been smokers at some time); and 58 percent in a third series were smokers. The histologic and clinical features of pulmonary histiocytosis X are discussed. The majority of patients, who are in the third and fourth decades of life, present with nonspecific respiratory complaints and bilateral reticulonodular infiltrates that can be seen on chest radiographs. The histologic lesions of pulmonary histiocytosis X are scattered, discrete nodules that frequently center on small airways. The initially cellular nodules are replaced progressively by fibrous tissue. The cell infiltrates in active lesions contain variable numbers of eosinophils and histiocytes resembling Langerhans cells of the epidermis (Hx cells). In most patients the disease resolves or stablizes, leaving few, if any, significant residues. A minority of patients develop progressive pulmonary disease that is ultimately fatal. Smokers' bronchiolitis and the "pseudodesquamative interstitial pneumonia reaction" may overshadow characteristic lesions of histiocytosis X and lead to misdiagnosis. (Auth. Abs. Mod.)

84–0468. Denjean, A.; Matran, R.; Mathieu, M.; Cerrina, J.; Duroux, P.; Lockhart, A. **Bronchial Response to Hyperventilation of Dry Air at Room Temperature in Normals and Asthmatics.** *Clinical Respiratory Physiology* 19(5): 477-482, 1983.

Results show that voluntary isocapnic hyperventilation of dry air at ambient temperature causes a mild, though significant, airway obstruction in normal nonsmokers as well as in current smokers. Because the fall in maximum expiratory flow at 50 percent of forced vital capacity (MEF$_{50}$) in normal smokers did not differ from that in normal nonsmokers, results of both subgroups were pooled. Bronchial effects of three levels of voluntary isocapnic hyperventilation of dry air at room temperature (20 to 22°C) were studied in 18 normal, nonatopic subjects (13 nonsmokers, 5 smokers) and in 25 nonperennial asthmatics who were asymptomatic and who had mild airway obstruction at the time of the study. The bronchial response was assessed using maximal expiratory flow (MEF) volume curves obtained before and 1, 5, 10, and 15 min after the 5 min hyperventilation challenge. In normal subjects, there was a minimal though significant ($p < 0.001$) fall in MEF that did not increase with the level of hyperventilation and was not accompanied by a fall in forced vital capacity (FVC). The bronchial response of asthmatics differed from that in normal subjects: the fall in MEF was significantly greater, associated with a significant fall in FVC, and increased with the level of hyperventilation. Results suggest that isocapnic voluntary hyperventilation of dry air at room temperature is a highly satisfactory screening test to detect bronchial hyperreactivity. (Auth. Abs. Mod.)

84–0469. Eisen, E. A.; Wegman, D. H.; Louis, T. A. **Effects of Selection in a Prospective Study of Forced Expiratory Volume in Vermont Granite Workers.** *American Review of Respiratory Disease* 128(4): 587-591, October 1983.

Longitudinal studies of forced expiratory volume in 1 sec (FEV$_1$) among groups of working men have found average rates of loss between -22 ml/year and -54 ml/year, depending on degree of smoking, while the average decline found in granite workers was -44 ml/year (-34 ml/year for nonsmokers). Workers who left the industry had an excessive mean rate of FEV$_1$ decline of -72 ml/year (-75 ml/year when retirees were excluded). In an examination of granite workers lost to followup during a 5-year study of pulmonary function, the application of a currently recommended repeatability criterion for pulmonary function tests (PFTs) was investigated by looking at different patterns of failing to satisfy the criterion (PFT failure) over multiple testing sessions. Workers with persistent PFT failure were found to have faster rates of FEV$_1$ decline than did subjects with none or only intermittent failures. In a regression analysis, job termination was found to be more strongly associated with a steep FEV$_1$ slope when it had been preceded by a pattern of persistent PFT failure. Cigarette smoking did not account for the accelerated rates of loss among terminated workers, implying that workers who continue in the granite sheds until retirement are not representative of all industry workers. (Auth. Abs. Mod.)

84–0470. Ekwo, E. E.; Weinberger, M. M.; Lachenbruch, P. A.; Huntley, W. H. **Relationship of Parental Smoking and Gas Cooking to Respiratory Disease in Children.** *Chest* 84(6): 662-668, December 1983.

Survey results of 1,355 children (aged 6 to 12 years) showed that the risk of hospitalization for respiratory illness before age 2 increased when at least one parent smoked ($p < 0.02$) or if cooking gas was used ($p < 0.001$). The occurrence of cough with colds increased significantly when one or both parents smoked ($p < 0.001$). Small but significant increases ($p < 0.05$) in the mean values of forced expiratory volume in 1 sec, the flow rate at 75 percent of the forced vital capacity, and the forced expiratory flow rate from 25 percent to 75 percent of the vital capacity were seen after administering inhaled isoproterenol to randomly selected children whose parents smoked ($n = 94$), but not among children whose parents did not smoke ($n = 89$). The same effects were not seen in association with gas cooking. Exposure of children during the first 2 years of life to gas cooking or cigarette smoking appears to be associated with an increased risk of hospitalization for respiratory illness, and cigarette smoking appears to be associated with a more consistent response to inhaled bronchodilator among 6- to 12-year-old children with no other history of chronic respiratory illness. (Auth. Abs.)

84–0471. Fernie, J. M.; Douglas, A.; Lamb, D.; Ruckley, V. A. **Right Ventricular Hypertrophy in a Group of Coalworkers.** *Thorax* 38(6): 436-442, June 1983.

The prevalence of right ventricular hypertrophy was low (15 percent) in the absence of progressive massive fibrosis, and was evident only in subjects who had smoked (170 of 204, 11 with unknown smoking history). Necropsy material from 215 coal workers (115 men with simple or no pneumoconiosis and 100 with progressive massive fibrosis) was examined. Right ventricular hypertrophy was considered to be present if the ratio of the weight of the left ventricle plus septum to that of the right ventricle was less than 2:1. Hypertrophy appeared to be related to emphysema or airways disease or both, and not to simple pneumoconiosis. In subjects with progressive massive fibrosis, the prevalence of right ventricular hypertrophy was higher (34 percent) and it was occasionally seen in nonsmokers. The prevalence increased with increasing size of lesion, and for any given lesion size, subjects with right ventricular hypertrophy had more panacinar emphysema than those without right ventricular hypertrophy. There was no relationship, however, between the extent of massive lesions or amount and type of emphysema and the degree of right ventricular hypertrophy. (Auth. Abs. Mod.)

84–0472. Greening, A. P.; Lowrie, D. B. **Extracellular Release of Hydrogen Peroxide by Human Alveolar Macrophages: The Relationship to Cigarette Smoking and Lower Respiratory Tract Infections.** *Clinical Science* 65(6): 661-664, December 1983.

Findings are consistent with the hypothesis that hydrogen peroxide (H_2O_2) of alveolar macrophage (AM) origin contributes to development of emphysema in smokers. AM were obtained by bronchoalveolar lavage and adherence from 24 smokers and 17 nonsmokers. Smokers' AM released significantly more H_2O_2 (mean 3.83 nmol/hour/µg of DNA; standard error (SEM) 0.44) than those of nonsmokers (mean 2.33 nmol/hour/µg of DNA; SEM 0.40; $p < 0.05$). AM from donors with a recent lower respiratory tract infection released increased quantities of H_2O_2 (mean 5.22 nmol/hour/µg of DNA; SEM 0.72; $p < 0.01$), even when allowance was made for smoking habits. (Auth. Abs. Mod.)

84–0473. Holland, W. W. **Evidence for the Implication of Environmental Factors in the Aetiology of Chronic Bronchitis.** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(2): 130-137, 1983.

While the single most important harmful factor in the etiology of bronchitis is cigarette smoking, environmental factors play a significant, if smaller, role. Morbidity and mortality due to bronchitis have been higher in Britain than other European countries; the regional variation also suggests environmental influences. There is a marked seasonal variation in bronchitis prevalence due to the independent influences of air temperature and air pollution. The relationship between air pollution and bronchitis has been confirmed for both children and adults, and a distinction is made between acute short-term and chronic long-term effects. In recent years, attention has focused on pollution of the microclimate within buildings. Occupational and other social factors may also be of etiological importance. (Auth. Abs.)

84–0474. Hunninghake, G. W.; Crystal, R. G. **Cigarette Smoking and Lung Destruction.** *American Review of Respiratory Disease* 128(5): 833-838, November 1983.

Results demonstrated an increased number of neutrophils in the lungs of smokers compared with nonsmokers. Cell suspensions, isolated from bronchoalveolar lavage fluid (8 smokers, 8 nonsmokers) and from open lung biopsies (3 smokers, 3 nonsmokers) with normal lung parenchyma, and from open lung biopsies of 5 nonsmokers and 4 smokers with sarcoidosis, were evaluated for the presence of neutrophils. A significantly increased number of neutrophils was present in the lavage fluid and lung biopsies of both normal and sarcoid smokers compared with nonsmokers ($p < 0.01$, each comparison). Alveolar macrophages from smokers' lavage fluids released large quantities of a chemotactic factor for neutrophils; those from nonsmokers did not. In vitro exposure to cigarette smoke stimulated the nonsmokers' macrophages to release the chemotactic factor. It is suggested that this mechanism is responsible for attracting neutrophils to smokers' lungs. (Auth. Abs. Mod.)

84–0475. Kasuga, H.; Shirai, F.; Takezawa, Y.; Mikami, R.; Mase, T.; Yuki, M.; Shiraishi, T. **Sekimen Kojo Jugyo-In ni Okeru hai Kino Shoken—Sekimen Bakuro to Kitsuen no Eikyo ni Tsuite. [Lung Function Tests in Asbestos Workers—Effect of Asbestos Exposure and Smoking.]** *Japanese Journal of Thoracic Diseases* 21(2): 127-133, February 1983, Japanese.

In lung function tests, performed on 51 asbestos workers with no respiratory symptoms and no X-ray evidence of any lung problems, smokers showed lower values than nonsmokers for all asbestos exposure categories. Workers were divided into six groups according to smoking status and length of time from first exposure to asbestos dust. Vital capacity (VC) and forced expiratory volume in 1 sec (FEV_1) declined with increasing duration of asbestos exposure. Among workers exposed to asbestos for more than 6 years, flow volume curve measurements were also abnormally decreased. Asbestos exposure was unrelated to the carbon monoxide diffusion capacity-lung capacity ratio (D_{LCO}/V_A). In heavy smokers, however, D_{LCO}/V_A was significantly diminished. It is noted that both smokers and nonsmokers who began employment after installation of asbestos dust collection equipment in the ventilation system showed a relatively small Brinkman index compared with the larger index values of those exposed prior to equipment installation.

84–0476. Linn, W. S.; Shamoo, D. A.; Venet, T. G.; Spier, C. E.; Valencia, L. M.; Anzar, U. T.; Hackney, J. D. **Response to Ozone in Volunteers With Chronic Obstructive Pulmonary Disease.** *Archives of Environmental Health* 38(5): 278-283, September-October 1983.

While smoking did not interact significantly with ozone exposure or time factors, analysis showed a significantly higher overall percent saturation of hemoglobin in current smokers (n=15) than in ex-smokers (n=11), suggesting that more severely diseased individuals were more likely to have quit smoking. Twenty-eight volunteers with chronic obstructive pulmonary disease were exposed to 0.0, 0.18, and 0.25 ppm ozone in purified air for 1-hr periods with light intermittent exercise, with exposure conditions presented in random order at 1-month intervals. No statistically significant changes attributable to ozone were found in forced expiratory performance or percent oxyhemoglobin (measured near the beginning and end of each exposure). No ozone-related changes in clinical status were found by interviews that included the time for 1 week before to 1 week after each exposure, except that a moderate increase in lower respiratory symptoms was reported by nonsmokers (n=2) in 0.18 ppm exposures only. Thus, a slight decrement in hemoglobin saturation with ozone exposure (reported in two previous studies of chronic obstructive pulmonary disease subjects) may not be a common occurrence under typical ambient exposure conditions. (Auth. Abs. Mod.)

84–0477. Litomericky, S.; Virsik, K.; Kristufek, P.; Smolenova, L.; Slukova, J.; Belan, P.; Mateffyova, I. **Klinicko-Epidemiologicka Studia o Chronickej Bronchitide u Osob Starsich ako 65 Rokov. [Clinico-Epidemiological Study of Chronic Bronchitis in Subjects Over the Age of 65 Years.]** *Bratislavske Lekarske Listy* 80(3): 279-285, September 1983, Czech.

Prevalence of chronic bronchitis was several times higher among cigarette smokers of both sexes than among nonsmokers, and rates increased with the amount smoked. Of 392 subjects aged over 65 years, chronic respiratory disease was diagnosed in 31 percent of men and in 18 percent of women. Chronic bronchitis was found most often, with an occurrence rate of 26 percent in men and 11 percent in women. Compared with healthy, elderly subjects, patients with chronic bronchitis presented significantly lower values of PaO_2, SaO_2, and higher values of $PaCO_2$. Hypoxemic respiratory insufficiency was demonstrated in 31.3 percent and hypercapnic in 19.2 percent of patients with chronic bronchitis. Preventive measures and early treatment of this disease improved the prognosis for longevity and better quality of life. (Auth. Abs. Mod.)

84–0478. Lungarella, G.; Fonzi, L.; Ermini, G. **Abnormalities of Bronchial Cilia in Patients With Chronic Bronchitis. An Ultrastructural and Quantitative Analysis.** *Lung* 161(3): 147-156, May 1983.

The fine structure of bronchial cilia was examined in biopsies from 18 chronic bronchitis patients (12 smokers, 2 ex-smokers, 4 nonsmokers) and 6 nonsmoking controls. Abnormal cilia, noted in three of the control subjects and in all patients with chronic bronchitis, ranged from 0 to 6 percent in controls and from 8 to 28 percent in bronchitis patients. Some ciliary abnormalities with changes in the axonemal 9 + 2 pattern of organization were only detected in patients with chronic bronchitis. Compound cilia with two or more complete axonemes arranged parallel to one another, and giant cilia with increased amounts of cytoplasmic matrix, were observed in both control and diseased subjects. Analysis of variance showed no significant differences between the smoking and nonsmoking patients in mean number of abnormal cilia, although the differences between patients and controls were significant. The data suggest that the presence of atypical cilia of epigenetic origin contributes to the severe impairment of mucociliary transport associated with chronic bronchitis. (Auth. Abs. Mod.)

84–0479. Maintz, G.; Schneider, W. D.; Rebohle, E. **Zur Interaktion von Arbeitsbedingten Noxen und Tabakrauchen. [The Interaction of Occupationally Conditioned Noxae and Smoking of Tobacco.]** *Zeitschrift fur die Gesamte Hygiene* 29(6): 320-324, 1983, German.

Few significant correlations were found between the effects of smoking and occupational exposures on heart and lung function among welders, wire varnishers, and carbon disulfide-exposed workers. However, the number of subjects was small, and the great majority were smokers. Also, the few nonsmokers included a high proportion of subjects disposed toward obstructive lung diseases, indicating an excess of these workers in groups not exposed to noxious substances. It is concluded that combined occupational and tobacco exposures are particularly harmful, but smoking is the more destructive element. Improvements in industrial working conditions are needed, but smoking cessation is emphasized as a more effective means of reducing health hazards.

84–0480. Mitic, M.; Sekulic, S.; Neradovic, L.; Havelka, M. **Utitsaj Duzhine Pushelja na Plucnu Funktsiju Mladikh Pushacha. [The Influence of Smoking on the Pulmonary**

Function in Young People.] *Serbian Archives of General Medicine* 109(11): 1465-1473, 1981, Serbo-Croatian.

Students at the University of Belgrade, Yugoslavia, who had smoked for more than 2 years showed significant declines in lung function. A group of 46 students who had smoked from 1 to 6 years, and 46 controls (all aged 18 years and asymptomatic) were evaluated for changes in respiratory function. As a whole, the smoker's lung function values were within normal limits when compared to the nonsmoker's. However, those who had smoked between 3 and 4 years showed significant changes in lung function, and those using cigarettes for more than 5 years manifested declines in respiratory function indicative of future chronic obstructive pulmonary disease.

84–0481. Moran, T. J. **Emphysema and Other Chronic Lung Disease in Textile Workers: An 18-Year Autopsy Study.** *Archives of Environmental Health* 38(5): 267-276, September-October 1983.

Since smoking histories were unavailable for many of the textile workers, smoking was not used as a factor in analysis, leaving some doubt as to the relationship between textile work and emphysema. Because smoking is so strongly associated with emphysema, it is possible that a marked difference in smoking habits between the textile workers and the control population could have masked a difference in emphysema incidence. A review of 2,895 consecutive autopsies, from 1962 to 1980, showed no significant differences in the prevalence of emphysema or other chronic lung disease between 282 active and retired employees of a cotton textile mill and the nontextile population. There was no statistical evidence that exposure to cotton dust, even after many years, produced emphysema, interstitial fibrosis, or cor pulmonale. The prevalence of emphysema in the series was highest in white males (22.0 percent), followed closely by black males (18.3 percent). In white females it was 7.5 percent, in black females, 5.5 percent. The prevalence in subjects under age 50 years was 4.5 percent; in the age group 60 to 64 years, 14.6 percent; and in subjects 65 years of age and older, 21.9 percent. A significant increase in the prevalence of emphysema occurred between the 1962 to 1969 period and the 1970 to 1980 period. (Auth. Abs. Mod.)

84–0482. Reiter, C. **Chromolipidspeicherung in Alveolarmakrophagen: Ein Agnoszierungsmerkmal fur Zigarettenraucher. [Storage of Chromolipids in Alveolar Macrophages: A Distinctive Mark for Cigarette Smokers.]** *Zeitschrift fur Rechtsmedizin* 91(1): 37-46, October 1983, German.

Histological and cytological examinations of 183 autopsies (113 smokers and 75 nonsmokers) established that cigarette smoke causes an increase and multiplication of alveolar macrophages that contain inclusions of ceroid-like pigment. Acute death by fire, delayed asphyxia, and putrefaction has no influence on the formation of these sudanophilic inclusions. The pigment is resistant to decomposition and lipid solvents. It is possible to identify cigarette smokers even months after burial by means of paraffin sections of lung tissue processed with fat-staining methods. (Auth. Abs.)

84–0483. Rom, W. N.; Lockey, J. E.; Bang, K. M.; Dewitt, C.; Johns, R. E., Jr. **Reversible Beryllium Sensitization in a Prospective Study of Beryllium Workers.** *Archives of Environmental Health* 38(5): 302-307, September-October 1983.

The effects of occupational beryllium exposure were independent of smoking-related effects in a 3-year prospective study of pulmonary changes consistent with chronic beryllium disease. Subjects were 59 employees of a surface mine and a process mill (12 smokers, 17 ex-smokers, and 30 nonsmokers). Blastogenic lymphocyte transformation (LT) to beryllium salts was observed in 15.9 percent of subjects in 1979, and in 8.2 percent in 1982. The annual decrement for forced vital capacity and forced expiratory volume in 1 sec was 107 and 81 ml in smokers compared with 60 and 37 ml in nonsmokers, respectively. However, the workers with positive LT did not show significantly reduced lung function, and multiple regression analysis revealed pack-years of smoking to be the only significant factor in reducing lung function measurements. No association between duration of employment and pulmonary function was found, and none of the chest radiographs had changes consistent with pneumoconiosis or beryllium disease.

84–0484. Rudnik, J.; Herman, S.; Pisiewicz, K.; Roszkowska, H. **Long-Term Effects on Health of Air Pollution in Children.** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(2): 177-191, 1983.

Passive smoking is considered a facilitating factor that increases the prevalence of chronic nonspecific respiratory disease (CNSRD) among children in a literature review that describes the role and relative importance of various endogenous and exogenous factors (classified as predisposing, provoking, and facilitating) in the development of CNSRD in children. Preliminary results of a longitudinal cohort study of 12,708 Polish children indicated that offspring of parents who both smoked and had respiratory disease showed a much higher incidence of morning cough, chronic cough, breathlessness, runny nose, and other symptoms of infection. The effect was much greater than expected from simple addition of the independent effects of passive smoking and parental respiratory disease, revealing synergistic interactions. Other significant factors discussed include a history of obstructive bronchitis in the first year of life, frequency of respiratory infections in early childhood, socioeconomic conditions including education of parents, day nursery and kindergarten attendance, and living in an area with high levels of air pollution. (Auth. Abs. Mod.)

84–0485. Tockman, M. S.; Becklake, M. R.; Clausen, J. L.; Cox, P. M., Jr.; Fontana, R. S.; O'Brien, R. J.; Permutt, S.; Petty, T. L.; Reed, C.; Reichman, L. B.; Stead, W. W. **American Thoracic Society. Medical Section of the American**

Lung Association. Screening for Adult Respiratory Disease. *American Review of Respiratory Disease* 128(4): 768-774, October 1983.

In a statement prepared by the Task Group on Screening for Adult Respiratory Disease, members consider the scientific justification of screening for (1) chronic airways obstruction, (2) asthma, (3) lung cancer, and (4) tuberculosis, by determining whether valid techniques permit early detection, and whether data exist to show that early detection results in a reduction of morbidity or mortality. Because there is no specific therapy for chronic airways obstruction (aside from smoking cessation), it is concluded that there is no basis for screening of the general population. Healthy, high-risk patients who should be considered for spirometric testing as part of a regular examination include heavy smokers, those exposed to occupational hazards, patients scheduled for thoracic or upper abdominal surgery (especially if obese or over 70 years of age), those on bronchodilator or beta-blocker medication, and those genetically predisposed to emphysema. There is also not sufficient evidence that screening for lung cancer by chest roentgenograms and sputum cytological examinations has led to a reduction in mortality. However, individual patients who request cancer-related checkups, especially patients in the high-risk categories (including middle-aged male cigarette smokers and those exposed to respiratory carcinogens) should be emphatically urged to stop smoking. Yet, only chest radiography and sputum cytology examinations have been shown to be capable of detecting presymptomatic bronchogenic carcinoma. If, in the physician's judgment, these might prove useful in cancer case recognition, there is no justification for withholding the tests. Specific recommendations are also provided for population screening and clinical case finding for asthma and tuberculosis. The statement was adopted by the American Thoracic Society in March 1983.

84-0486. Velluti, G.; Capelli, O.; Lusuardi, M.; Braghiroli, A.; Pellegrino, M.; Milanti, G.; Benedetti, L. **Bronchoalveolar Lavage in the Normal Lung. First of Three Parts: Protein, Enzymatic and Ionic Features.** *Respiration* 44(6): 403-410, November-December 1983.

Bronchoalveolar lavage was performed on 12 healthy volunteers (6 smokers and 6 nonsmokers), between 21 and 52 years of age, to define normal variability of certain biochemical, immunologic, enzymologic, and ionic parameters. Smoking exerted a significant influence on the recovery percentage of lavage effluents (with recovery less in smokers, 53 vs. 69 percent) and particularly on the concentration of immunoglobulins in the lavage liquids. Immunoglobulin G levels increased by about four times in smokers (1.05 vs. 0.26 mg/100 ml) and the immunoglobulin A by about three times (0.35 vs. 0.11 mg/100 ml). The other parameters studied did not differ significantly between groups. Rather than an alteration in the blood-alveolar barrier from smoking, the data suggest a real local overproduction of immunoglobulins of classes G and A induced by smoking. Moreover, the relatively slight individual oscillation in the values of the parameters studied in the two groups supports the possibility of employing them for diagnostic purposes in bronchopneumopathies. (Auth. Abs.)

84-0487. Weill, H. **Asbestos-Associated Diseases. Science, Public Policy, and Litigation.** *Chest* 84(5): 601-608, November 1983.

There appears to be a surfeit of discussion regarding the role of government in compensation for asbestos-exposed workers, yet a curious lack of interest in determining how many claimants are really ill and which have asbestos-attributable disease. It is noted in this review that although smoking does not affect risk of mesothelioma, very few cases of lung cancer occur in nonsmoking asbestos workers. No histologic type of this tumor, or location in the lung, can be linked to asbestos exposure. Also, smoking is the most prominent factor in the development of chronic airways obstruction, while it is unlikely that asbestos measurably contributes to obstructive pulmonary diseases. The influence of science on past policy decisions is discussed, in addition to an historical perspective of the problem, public responsibility in compensation and other economic issues, dose—response data, setting of standards, and the implications of these factors for future policy decisions.

84-0488. Weiss, S. T.; Tager, I. B.; Speizer, F. E. **Passive Smoking. Its Relationship to Respiratory Symptoms, Pulmonary Function and Nonspecific Bronchial Responsiveness.** *Chest* 84(6): 651-652, December 1983.

While data from a current study (*Chest* 84(6): 662-668, December 1983) apparently do not support an effect of parental smoking on either lung function or bronchial responsiveness, it is noted that the interrelationships between respiratory illness, parental smoking, and the atopic state are complex. The study did demonstrate a relationship between parental smoking and hospitalization for respiratory illness before the age of 2 years, and it thus appears parental smoking may contribute to respiratory symptoms and morbidity in young children. Another possible health consequence of passive smoke exposure in early life may be on maximally attained lung function in adulthood. While the effect may be small, it may be an indicator of those at risk of developing obstructive airways disease when exposed more directly to personal (e.g., cigarette smoke) or environmental agents in adult life, an hypothesis that is currently under investigation. See also 84-0470

84-0489. Weiss, T.; Dorow, P.; Felix, R. **Regional Mucociliary Removal of Inhaled Particles in Smokers With Small Airways Disease.** *Respiration* 44(5): 338-345, September-October 1983.

In contrast with findings in 20 nonsmokers without evidence of small airways dysfunction, significant mucociliary clearance delay occurred in the peripheral and upper pulmonary regions in 30 asymptomatic smokers. Quantitative radioaerosol lung imaging was performed in smokers selected on the basis of abnormal findings in pulmonary function tests of small airways. Regional clearance rates of inhaled ^{99m}Tc-labeled human serum albumin minimicrospheres were determined over upper, middle, lower, central, and peripheral anterior lung zones. An aerosol distribution index (ADI) measures the homogeneity of distribution of inhaled particles in the lung. A significant difference in the mean values of ADI was found, indicating an abnormal uneven radioaerosol distribution pattern in the smokers with small airways disease. (Auth. Abs.)

84–0490. Wright, J. L.; Lawson, L.; Pare, P. D.; Hooper, R. O.; Peretz, D. I.; Nelems, J. M.; Schulzer, M.; Hogg, J. C. **The Structure and Function of the Pulmonary Vasculature in Mild Chronic Obstructive Pulmonary Disease. The Effect of Oxygen and Exercise.** *American Review of Respiratory Disease* 128(4): 702-707, October 1983.

Possible mechanisms of pulmonary hypertension were investigated in smokers with mild or moderate emphysema ($n=13$) and nonsmoking controls, all requiring pulmonary resection. All subjects performed pulmonary function tests and smokers underwent right heart catheterization preoperatively. The surgical specimens of the lung were analyzed with respect to the severity of the emphysema, the severity of the airways disease, and the hypertensive changes in the small pulmonary arteries and arterioles. Among smokers, pulmonary artery (Ppa) and pulmonary artery wedge (Ppaw) pressures and cardiac output were normal at rest. However, patients with more severe disease showed greater increases in Ppa and Ppaw with exercise than did patients with minimal or no disease. Oxygen breathing had no effect at rest but lowered Ppa and Ppaw during exercise in patients with more severe disease. Histologic studies showed that greater degrees of emphysema and small airways disease were associated with hypertensive changes in the pulmonary vasculature. Compared with nonsmoking controls, the smokers had a significatly increased percentage of arterial intima and decreased percentage of adventitia. The relative proportion of media did not change. Adventitial thickness was directly related to Ppaw. It is concluded that patients with moderate obstructive lung disease have elevated pulmonary vascular pressures during exercise and structural changes in the pulmonary arteries consistent with pulmonary hypertension when compared with patients with minimal or no disease. Although there are several possible explanations for the increased vascular pressures seen with exercise, it is suggested that gas trapping leading to increased alveolar and pleural pressures, with oxygen breathing, prevents this response by shortening the time constants of the peripheral lung units. (Auth. Abs. Mod.)

See also, **84–0371, 84–0412, 84–0420, 84–0453, 84–0504**

CARDIOVASCULAR DISEASES

84–0491. Aronow, W. S. **Carbon Monoxide and Cardiovascular Disease.** *Comprehensive Therapy* 9(10): 21-26, October 1983.

Carbon monoxide exposure from heavy smoking or heavy atmospheric carbon monoxide pollution contributes to the increase in nonfatal and fatal myocardial infarction and sudden death from coronary artery disease through carboxyhemoglobin (1) interfering with myocardial oxygen delivery, (2) depressing the pump performance of the heart muscle, (3) reducing the threshold for ventricular fibrillation, and (4) increasing platelet stickiness, thereby increasing a thrombotic tendency. In addition, data indicate that high smoking or occupational exposure to carbon monoxide contributes to pathogenesis of atherosclerotic disorders, aggravates angina pectoris and intermittent claudication of the calf or thigh, and increases myocardial ischemia in patients with clinical and subclinical coronary artery disease. Patients with chronic obstructive lung disease, cerebrovascular disease, and anemia are also more susceptible to adverse health effects from low levels of carbon monoxide exposure. Pregnant women, fetuses, and newborn infants are particularly sensitive. (Auth. Abs. Mod.)

84–0492. Borland, C.; Chamberlain, A.; Higenbottam, T. W.; Shipley, M. J.; Rose, G. **Carbon Monoxide Yield of Cigarettes and Its Relation to Cardiorespiratory Disease.** *British Medical Journal* 287(6405): 1583-1586, November 26, 1983.

Estimates of the carbon monoxide yield of smokers' cigarettes were obtained for 4,910 subjects (68 percent of all smokers) in the Whitehall study of men aged 40 to 64. In the 10 years following initial examination, 635 men died. When men smoking cigarettes with high carbon monoxide yield were compared with those smoking cigarettes with a low yield (after adjusting for age, employment grade, amount smoked, and tar yield), the risk of death was 32 percent lower for coronary heart disease, 49 percent higher for lung cancer, and 10 percent lower for total mortality; these differences were not statistically significant. Among men who said they inhaled, the risk of fatal coronary heart disease was 51 percent lower in the high carbon monoxide group ($p<0.01$), while the risk of lung cancer was 75 percent higher. Results provide no evidence that a smoker can reduce the risk of death by smoking a brand with a low carbon monoxide yield. (Auth. Abs. Mod.)

84-0493. Brown, T. E.; Myles, W. S.; Allen, C. L. **The Relationship Between Aerobic Fitness and Certain Cardiovascular Risk Factors.** *Aviation Space and Environmental Medicine* 54(6): 543-547, June 1983.

Incidence of smoking declined slightly as fitness category improved in 2,501 Canadian servicemen (aged 18 to 50 years) compared for aerobic fitness and the incidence of cardiovascular disease risk factors. Subjects were divided into three age groups, and subdivided into four fitness categories. This trend was much more evident in the two older groups, but was entirely absent among those subjects under 30 years of age. Aerobic power was predicted from heart rate measured during submaximal bicycle exercise. Risk factors measured included serum cholesterol, serum triglycerides, blood pressure, and body fat; smoking data were obtained by questionnaire. An inverse relationship, demonstrated between fitness category and the mean values for all measured risk factors, was most evident in the older subjects; however, it is significant that it was also present in men under 30 years of age. A similar inverse relationship was demonstrated for all age groups between fitness category and the percent of subjects considered to have elevated cholesterol, triglycerides, blood pressure, or skinfold thickness. It is suggested that participation in a program to improve physical fitness may lead individuals to modify other lifestyle components. (Auth. Abs. Mod.)

84-0494. Burch, P.R.J. **Diet and Ischaemic Heart Disease. (Letter).** *Lancet* 2(8352): 743, September 24, 1983.

While the relative risk of ischemic heart disease (IHD) in heavy smokers and persons with hypertension and hypercholesteremia is high (3 to 6) in those aged 40 to 44, it declines rapidly to 1 to 2 in those aged 60 to 64. A complex blend of changes in IHD risk factors with age, sex, and time could account for mortality trends, but it is suggested that the decline in mortality may have resulted from the decreasing impact of a ubiquitous precipitating factor such as a microorganism. Such a theory would provide a better explanation for seasonal fluctuations in mortality and the age-dependence of relative risks. The associations of various risk factors with IHD could then be interpreted on a genetic basis. It would thus be unlikely that hypercholesterolemia and hypertension have any appreciable causal action.

84-0495. Castelli, W. P. **Cardiovascular Disease and Multifactorial Risk: Challenge of the 1980s.** *American Heart Journal* 106(5, Part 2): 1191-1200, November 1983.

Cigarette smoking continues to be one of the three most significant coronary heart disease (CHD) risk factors in persons under age 60. However, the Framingham Heart Study indicates that risk declines to nonsmokers' levels within 1 year of quitting for CHD and within 10 to 15 years for lung cancer. While underweight persons were previously believed to be at high mortality risk (some insurance companies considered raising their recommended ideal weight levels), data show that death in underweight men occurs primarily from cancer, especially smoking-associated lung cancer (82 percent of men in the lowest weight group are smokers). Nonsmoking underweight males have the lowest cardiovascular and total death rate. Physicians are challenged to adopt a genuinely multifactorial approach to CHD risk factor management. Two practical tools are described: (1) a precise number evaluation system that calculates risk using five parameters (total cholesterol, systolic blood pressure, absence or presence of diabetes mellitus, electrocardiographic evidence of left ventricular hypertrophy, and cigarette smoking) and a multiplier for high density lipoprotein cholesterol; and (2) a categorical checklist that provides cutoff values for all risk factors as an aid in identifying those patients whose overall middle-value profile places them at high risk. Rates of morbidity from heart attack in relation to cigarette smoking are provided. The natural history of the atherosclerotic cardiovascular diseases is reviewed in terms of the risk factor parameters in the Framingham Heart Study population over a period of 18 years. (Auth. Abs. Mod.)

84-0496. Chen, H.; Zhuang, H.; Han, Q. **Serum High Density Lipoprotein Cholesterol and Factors Influencing Its Level in Healthy Chinese.** *Atherosclerosis* 48(1): 71-79, July 1983.

No statistically significant differences were detected in mean serum high density lipoprotein cholesterol (HDL-C) concentrations in a subgroup of 242 male smokers (20 cigarettes/day) compared with 149 male nonsmokers. Serum HDL-C levels were analyzed in 2,399 healthy urban inhabitants (newborn to 78 years of age) in Shanghai, China. Overall results showed that frequency distribution of serum HDL-C levels in most groups was approximately normal after age 20. Serum HDL-C levels and HDL-C total cholesterol ratio varied with age and sex among healthy subjects. The mean values of HDL-C for people over 30 years of age were 58.7±11.2 mg/100 ml for males and 62.5±12.2 mg/100 ml for females (statistically significant). Mean values of serum HDL-C were found to be 40.9±6.0 mg/100 ml and 40.7±7.9 mg/100 in male and female neonates, respectively. These figures are similar to those reported from Western countries and Japan. The serum HDL-C level rose steadily with age, and was more marked in females than males before age 30. However, the values became closer at age 60, then began to decrease in both sexes. Serum HDL-C levels varied markedly with body weight, correlating negatively with the weight/height index. No statistically significant relationship was found between alcohol consumption and serum HDL-C levels. The high level of HDL-C seemed a result of environmental factors and is, possibly, related to the lower incidence of coronary heart disease in China. (Auth. Abs. Mod.)

84-0497. Fuller, J. H.; Shipley, M. J.; Rose, G.; Jarrett, R. J.; Keen, H. **Mortality From Coronary Heart Disease and Stroke in Relation to Degree of Glycaemia: The Whitehall Study.** *British Medical Journal* 287(6396): 867-870, September 24, 1983.

Smokers, but not ex-smokers, had significantly increased risk of death from coronary heart disease (CHD), but not from stroke, in the Whitehall study of 18,403 male civil servants aged 40 to 64 years. The 10-year mortality rates from CHD and stroke showed a nonlinear relation to 2-hour blood glucose values, with a significantly increased risk for glucose-intolerant subjects and for diabetics. Multiple logistic analysis showed that between 50 and 75 percent of the relative risks for deaths from CHD and stroke were unexplained by differences in age, blood pressure, obesity, smoking, cholesterol concentration, and electrocardiographic abnormalities. Within the glucose-intolerant and diabetic groups, the risk factors most strongly related to subsequent death from CHD were age and blood pressure, with less consistent relationships for smoking, cholesterol concentration, and obesity. Age-adjusted 10-year mortality of normoglycemic subjects was 5.11 for smokers, 2.73 for ex-smokers, and 2.03 for nonsmokers. Data for the glucose-intolerant and diabetic groups varied widely. (Auth. Abs. Mod.)

84–0498. Giagnoni, E.; Secchi, M. B.; Wu, S. C.; Morabito, A.; Oltrona, L.; Mancarella, S.; Volpin, N.; Fossa, L.; Bettazzi, L.; Arangio, G.; Sachero, A.; Folli, G. **Prognostic Value of Exercise EKG Testing in Asymptomatic Normotensive Subjects. A Prospective Matched Study.** *New England Journal of Medicine* 309(18): 1085-1089, November 3, 1983.

Smoking, age, systolic blood pressure, coronary risk index, and exercise electrocardiographic (EKG) tests were found to be of value in predicting coronary events. Cox's regression model was used to evaluate data from a study of 135 asymptomatic normotensive subjects with exercise-induced ST ischemic depression of 1 mm or more, compared with 379 controls. At least two controls with negative responses on the exercise EKG test were selected for each case and were matched for age, sex, work, community, and index of coronary risk factors. The coronary events considered as end points were angina pectoris, myocardial infarction, and sudden death. After a median followup period of 6 years for cases and 6.4 years for controls, the relative risk was 5.55 (95 percent confidence limits, range 2.75 to 11.22). Coronary events occurred significantly earlier in the cases than in the controls. Data also suggest that the exercise EKG response is a particularly good prognostic indicator for myocardial infarction. (Auth. Abs. Mod.)

84–0499. Green, M. A.; Egle, J. L., Jr. **The Effects of Acetaldehyde and Acrolein on Blood Pressure in Guanethidine-Pretreated Hypertensive Rats.** *Toxicology and Applied Pharmacology* 69(1): 29-36, June 15, 1983.

A significant interaction between the antihypertensive agent guanethidine and two aliphatic aldehydes (acetaldehyde and acrolein, components of the vapor phase of cigarette smoke) was demonstrated in spontaneously hypertensive rats (SHRs). Acetaldehyde, when administered i.v. to acutely guanethidine-pretreated (15 mg/kg) SHRs under urethane anesthesia, caused a potentiated pressor response in the dose range of 3 to 40 mg/kg. When administered i.v. to chronically guanethidine-pretreated SHRs, a pressor response was noted at low doses and a depressor response at high doses. Acrolein (0.05 to 0.5 mg/kg) produced a pressor response at low doses and a depressor response at high doses in both acutely and chronically guanethidine-pretreated SHRs. Pressor responses, particularly to acetaldehyde, may be due to an enlarged tyramine-releasable pool, hyperreactivity of α adrenergic receptors of SHRs, or guanethidine inhibition of norepinephrine reuptake. Depressor responses to high doses of aldehydes may be attributed to vagal stimulation or direct vasodilation. It is concluded that the interaction between the aldehydes and guanethidine may have implications for those being treated for hypertension with guanethidine while being exposed to acetaldehyde and related compounds from ethanol and tobacco smoke. (Auth. Abs. Mod.)

84–0500. Haigh, N. Z.; Salz, K. M.; Chase, G. A.; Jenkins, J. A.; Bachorik, P. S.; Kwiterovich, P. O., Jr. **The East Baltimore Study: The Relationship of Lipids and Lipoproteins to Selected Cardiovascular Risk Factors in an Inner City Black Adult Population.** *American Journal of Clinical Nutrition* 38(2): 320-326, August 1983.

Smoking habits did not account for the relatively low total cholesterol and low density lipoprotein cholesterol (LDLC) levels in inner-city men. Plasma lipid and lipoprotein levels were analyzed in black adults from East Baltimore, Maryland (24 males, 45 females), with respect to smoking, dietary intake, alcohol consumption, obesity, physical activity, and hormone use. Smoking accounted for 4.27 percent of variance in triglycerides and 0.05 of variance in cholesterol levels among the women. Mean lipid and lipoprotein levels were similar in the men and women. However, the men's total cholesterol (167 mg/dl) and LDLC (94 mg/dl) levels were lower than those of adult blacks in other studies, while the levels of the East Baltimore women were similar to those in other studies. For women, body mass index and high density lipoprotein cholesterol were negatively correlated ($p < 0.01$), while triglycerides and oral contraceptive use were positively correlated ($p < 0.01$). None of the factors studied explained the relatively low total cholesterol and LDLC levels among the men. (Auth. Abs. Mod.)

84–0501. Henry, J. P.; Stephens, P. M. **Caffeine, Stress, and Cardiomyopathy.** *Journal of the South Carolina Medical Association* 79(10): 545-548, October 1983.

Results suggest that the caffeine habit should be considered in addition to smoking, high blood pressure, and type A behavior as a risk factor for sudden death in persons diagnosed as having coronary heart disease. An investigation of the effects of caffeine in water (and tea and coffee) in mice living as peaceful siblings in boxes and as agitated members of a competitive social system, showed that even the addition of 3.3 mg caffeine/day/kg mouse body weight (equivalent of someone drinking two 250 cc cups of strong coffee) intensified the psychosocial stresses on male mice in the presence of females. Because tobacco smoking so often goes with coffee drinking, it has been difficult to disentangle the effects of nicotine from caffeine as risk factors for heart disease and sudden cardiac death. However, the behavioral risk factors for coronary heart disease (anxiety, irritability, depression, and increased hostility) can be found in those with the caffeine habit. In mice, the caffeine-induced enhancement of competitive social stimulation of neuroendocrine arousal resulted in a further increase of plasma renin and corticosterone levels and increase in the already elevated blood pressure and adrenal weight. It is concluded that these changes, together with the further acceleration of mortality and increased incidence of cardiac fibrosis and interstitial nephritis, indicate that chronic consumption of caffeinated beverages adds to the risk of psychosocial stress.

84–0502. Jugdutt, B. I.; Stevens, G. F.; Zacks, D. J.; Lee, S.J.K.; Taylor, R. F. **Myocardial Infarction, Oral Contraception, Cigarette Smoking, and Coronary Artery Spasm in Young Women.** *American Heart Journal* 106(4): 757-761, October 1983.

Coronary spasm is thought to play an important role in the pathogenesis of myocardial infarct (MI) in young menstruating women who smoke and take oral contraceptives (OCs), and who are likely to have normal coronary arteries. To test the hypothesis that the combination of smoking and OC use provides a strong stimulus for platelet aggregation, coronary spasm, and thrombosis, 65 women with atypical chest pain and normal coronary arteriograms were studied: 20 were smokers, 16 were menstruating (15 smokers), 9 took OCs, and 2 developed MI. It is thought that smoking may have precipitated spasm via nicotine-induced release of norepinephrine and diminished prostacyclin/thromboxane A_2 ratio, and the combination of smoking and estrogens causing a cycle of platelet aggregation, vasoconstriction, and thrombosis, leading to MI in at least one patient.

84–0503. Kaufman, D. W. **Constituents of Cigarette Smoke and Cardiovascular Disease.** *New York State Journal of Medicine* 83(13): 1267-1268, December 1983.

Although the risk of fatal and nonfatal myocardial infarction was higher in smokers than nonsmokers, the magnitude of risk was not correlated with carbon monoxide and nicotine levels of cigarette brands in three studies. This implies that either (1) nicotine and carbon monoxide are not the constituents in smoke that contribute to coronary disease; or (2) that smokers are absorbing more of these compounds than indicated by Federal Trade Commission-established ratings. A growing body of data suggests the latter reason may be valid, since several studies have shown that smokers of low-nicotine cigarettes take in as much nicotine as smokers of higher-rated products, and also that carboxyhemoglobin levels do not correlate with cigarette yields. Although it is possible another substance may be responsible, other biologic evidence indicates that nicotine and carbon monoxide are the risk-increasing factors. Smokers of low-yield brands should be warned that these products do not reduce risk of coronary heart disease.

84–0504. Keith, R. E.; Mossholder, S. B. **Vitamin C and Acute Physiological Responses to Cigarette Smoking.** *Nutrition Research* 3(5): 653-661, September-October 1983.

Results suggest a dose—response effect of ascorbic acid (AA) administration on heart rate (HR) and blood pressure (BP) following smoking. AA and sugar placebos (S) were administered double-blind to 62 male and female subjects aged 18 to 24. Groups received either a single dose of 400 mg or 1200 mg AA or S prior to testing. During testing, subjects were required to smoke two cigarettes within 10 minutes. Before and after smoking, values were obtained for resting HR, systolic and diastolic BP, forced vital capacity, forced expiratory volume in 1 sec, and maximal flow rate. Smoking adversely affected all parameters measured. While 400 mg AA resulted in a significantly smaller increase in systolic BP following smoking, 1200 mg AA resulted in significantly larger increases in diastolic BP and HR. Effects of AA on HR and BP were more pronounced in females than in males. No differences in lung function were seen with AA supplementation. (Auth. Abs. Mod.)

84–0505. Klein, L. W.; Gorlin, R. **The Systemic and Coronary Hemodynamic Response to Cigarette Smoking.** *New York State Journal of Medicine* 83(13): 1264-1266, December 1983.

Cigarette smoke exerts an influence on the cardiovascular system by increasing blood pressure, heart rate, cardiac output, myocardial contractile force and contraction velocity, and by changes in stroke volume. Smoking also affects coronary blood flow. As a result of the hemodynamic changes, myocardial oxygen demand increases while carboxyhemoglobin levels rise, limiting myocardial oxygen supply. In response, coronary blood flow also increases, although myocardial oxygen extraction is submaximal under the greater exertion. If the coronary vasculature cannot compensate for the increased workload, myocardial ischemia and more severe angina may result. The roles of nicotine and carbon monoxide and their consequences to heart disease patients are also discussed.

84–0506. Kubota, K.; Yamaguchi, T.; Abe, Y.; Fujiwara, T.; Hatazawa, J.; Matsuzawa, T. **Effects of Smoking on Regional Cerebral Blood Flow in Neurologically Normal Subjects.** *Stroke* 14(5): 720-724, September-October 1983.

Chronic smoking reduces regional cerebral blood flow (CBF) in neurologically normal subjects, probably due to advanced atherosclerosis, which produces vascular narrowing and raised resistance in cerebral blood vessels. Smoking effects on CBF and on serum lipids and lipoprotein levels were studied in 111 neurologically normal subjects (37 smokers and 74 nonsmokers). Smokers' mean smoking index (number of cigarettes/day

X number of years smoking) was 760. In the nonsmoking group, 62 of the 74 subjects had never smoked; the mean smoking index of nonsmokers was 17. In males, CBF was significantly lower in smokers than in nonsmokers (mean CBF, 12.5 percent lower in smokers, $p<0.001$). Increased reduction of CBF with advancing age was also observed. Compared with nonsmokers, CBF in smokers was found to be significantly lower than the expected age-matched value. Serum high density lipoprotein cholesterol values were significantly lower, and total cholesterol levels were significantly higher in smokers than in nonsmokers. (Auth. Abs. Mod.)

84–0507. Lang, T.; Bureau, J. F.; Degoulet, P.; Salah, H.; Benattar, C. **Blood Pressure, Coffee, Tea and Tobacco Consumption: An Epidemiological Study in Algiers.** *European Heart Journal* 4: 602-607, 1983.

Systolic blood pressure was negatively and significantly correlated with cigarette smoking in an investigation of the interactions between coffee drinking, tobacco use, blood pressure (BP), age, and body mass index (BMI). These factors were analyzed in a cross-sectional epidemiologic study of 1,098 men and 393 women from the Algiers district. Diastolic blood pressure (DBP) was positively and significantly associated with coffee consumption ($p<0.001$) and negatively and significantly with cigarette consumption ($p<0.001$). These associations remained significant after multivariate analysis, including sex, age, BMI, physical exertion at work, athletic activity, rural vs. urban residency, tobacco, tea, and coffee consumption. Cigarette smoking was positively associated with coffee consumption ($p<0.001$). In men, the association between coffee consumption and DBP was significant only after adjustment for smoking; among women, there was a low percentage of smokers, and this association was significant without such adjustment. There was no significant positive correlation between BP and tobacco chewing. It is suggested that cigarette consumption might mask the association between coffee consumption and BP. The contradictory interactions observed between BP, smoking, and coffee consumption might help to explain the conflicting reports regarding the association between coffee consumption and ischemic heart disease.

84–0508. Leren, P.; Helgeland, A.; Hjermann, I.; Holme, I. **The Oslo Study: CHD Risk Factors, Socioeconomic Influences, and Intervention.** *American Heart Journal* 106(5, Part 2): 1200-1206, November 1983.

Results of the large-scale, 10-year Oslo study relate cause-specific mortality to the major coronary heart disease (CHD) risk parameters of serum cholesterol, cigarette smoking, and blood pressure, indicating that risk factors (especially cigarette smoking and serum cholesterol) are highest among the lowest socioeconomic classes. Data also associated workers on lower occupational—educational levels (i.e., transport workers, longshoremen, construction workers, wholesale tradesmen, industrial workers) with higher prevalence of smoking and serum cholesterol, and showed that more highly educated subjects tended to have lower cholesterol levels and rarely smoked (their total risk was one-third that of the lower occupational—educational group). Morbidity from myocardial infarction and total cardiovascular incidence decreased with lowered blood lipid levels and smoking cessation in one of two controlled intervention trials described (one in healthy, normotensive men at high CHD risk, the other in healthy men with mild hypertension). In the first trial group, 604 and 628 men were randomly assigned to an intervention and a control group, respectively. At the start of the trial, 80 percent of the men in both groups smoked cigarettes daily. Mean tobacco consumption decreased 45 percent more in the intervention group than in the control group; 25 percent of the intervention group and 17 percent of the control group stopped smoking entirely. At the end of the observation period, the incidence of myocardial infarct was 47 percent lower in the intervention group than in the control group ($p=0.028$). Total cardiovascular incidence was also lower in the intervention group. There was no difference in total cardiovascular incidence as a result of antihypertensive therapy in the other trial. The effects on blood lipids of six commonly used single antihypertensive drug preparations and five combination preparations were examined. The study concluded that the beneficial lipid-lowering effect of the alpha-adrenergic blocker prazosin made it the antihypertensive drug of choice. (Auth. Abs. Mod.)

84–0509. Marshall, M. **Beziehungen Zwischen Arterioskleroserisikofaktoren 1. Ordnung und Hamorheologischen Parametern. [Correlations Between the Main Risk Factors for Arteriosclerosis and Hemorrheological Parameters.]** *Fortschritte der Medizin* 101(38): 1716-1726, October 1983, German.

Nearly all studies of blood component changes in subjects with risk factors for arteriosclerosis have indicated that hyperlipidemia, hypertension, and/or smoking are accompanied by (1) increased blood and plasma viscosity (increased blood viscosity and hematocrit in smokers); (2) diminished flexibility and filterability of red blood cells; and (3) hyperaggregation and shortened lifespan of platelets. Risk factors are additive in the case of blood filterability, producing acute or subacute blood component changes before angiopathy can be experimentally developed. Thus, risk factors and blood component changes may be consecutive factors or important parallel factors in atherogenesis. (Auth. Abs. Mod.)

84–0510. Martin, C. A.; Thompson, P. L.; Armstrong, B. K.; Hobbs, M.S.T.; De Klerk, N. **Long-Term Prognosis After Recovery From Myocardial Infarction: A Nine Year Follow-Up of the Perth Coronary Register.** *Circulation* 68(5): 961-969, November 1983.

Smoking status at time of myocardial infarction (MI) showed no association with long-term prognosis among 1,078 patients on the 1971 Perth Coronary Register who were followed up for 9 years. Of the patients studied, 77 percent survived the first 24 hours and 62 percent the first 28 days (0.3 percent were lost to followup). For the 666 patients who were alive at 28 days, the crude 1-, 5-, and 9-year survival rates were 88, 67, and 52 percent, respectively. The relationship between 54 variables and the survival of patients alive 28 days after MI was examined by life-table methods and the log rank test, and by fitting a proportional hazards model to the data. The important prognostic factors were age, sex, past history of MI, stroke, diabetes and hypertension, tachycardia at presentation, hypotension at presentation, and the occurrence of arrhythmias as short-term complications. Initial smoking habits, Quetelet index, and angina were not significant prognostic factors. The most appropriate mathematical description of the joint effects of the prognostic factors was a multiplicative model with no interaction. (Auth. Abs. Mod.)

84–0511. Mikhailidis, D. P.; Barradas, M. A.; Jeremy, J. Y.; Dandona, P. **Cigarette Smoking Inhibits Prostacyclin Formation. (Letter).** *Lancet* 2(8350): 627-628, September 10, 1983.

A rise in nonesterified fatty acid (NEFA) concentration, which may contribute to platelet activation in smokers through described effects, is suggested as an additional mechanism to consider regarding findings of a previous report (*Lancet* 1(8336): 1248-1250, June 4, 1982) on the relationship between vascular disease and reduced vascular prostacyclin (PGI_2) production in heavy smokers. Relevant previous research is discussed. It is suggested that smokers produce less PGI_2 than nonsmokers because (1) chronic smoking results in endothelial cell damage, which may be cumulative; and (2) chronic smokers may produce a greater elevation in serum NEFA concentrations than nonsmokers in response to nicotine inhalation. See also 83-1568.

84–0512. Nicholson, J. P.; Alderman, M. H.; Pickering, T. G.; Teichman, S. L.; Sos, T. A.; Laragh, J. H. **Cigarette Smoking and Renovascular Hypertension.** *Lancet* 2(8353): 765-766, October 1, 1983.

Results of a retrospective cohort study comparing 71 patients with documented renovascular hypertension and 308 age-matched controls with essential hypertension showed that 94 percent (30 of 32) of men and 74 percent (29 of 39) of women with renal artery stenosis had smoked cigarettes, compared with only 43 percent (64 of 150) of men and 41 percent (65 of 158) of women in the control group. This striking relationship was true for both patients with fibromuscular disease (71 percent smokers) and patients with atherosclerotic lesions (88 percent smokers). All renal artery stenosis groups had significantly higher systolic and diastolic blood pressures than the relevant control group. When the groups were stratified according to blood pressure, there were significantly more smokers in the renal artery stenosis group at every level of blood pressure. (Auth. Abs. Mod.)

84–0513. Pero, R. W.; Bryngelsson, C.; Bryngelsson, T.; Lindholm, L.; Schersten, B.; Trell, E.; Janzon, L.; Franzen, J.; Johansson, B. W. **The Effects of Smoking, Hypertension and Cardiovascular Disease on the Estimation of Unscheduled DNA Synthesis and Covalent Binding to DNA Induced by *N*-Acetoxy-2-acetylaminofluorene in Resting Human Mononuclear Leukocytes.** *Atherosclerosis* 48(2): 119-129, August 1983.

The levels of *N*-acetoxy-2-acetylaminofluorene (NA-AAF)-induced unscheduled DNA synthesis (UDS) and of NA-AAF binding to DNA were determined in resting mononuclear leukocytes from individuals with various smoking habits, myocardial infarct patients, and subjects diagnosed for hypertension. Age-matched and blood pressure-controlled smokers ($n=99$) had significantly elevated levels of NA-AAF-induced UDS and NA-AAF binding to DNA when compared with nonsmokers ($n=75$). Myocardial infarct patients without any history of risk factors and hypertensives with normalized blood pressure were not significantly different from matched controls when assessed by the NA-AAF method. The results support the theory that increased mutagen sensitivity is associated with smoking and high blood pressure, but not with cardiovascular disease itself, via some mechanism of genetic selection. (Auth. Abs. Mod.)

84–0514. Proudfit, W. J.; Bruschke, A.V.G.; MacMillan, J. P.; Williams, G. W.; Sones, F. M., Jr. **Fifteen Year Survival Study of Patients With Obstructive Coronary Artery Disease.** *Circulation* 68(5): 986-997, November 1983.

Survival rates were examined in 598 patients (175 nonsmokers, 345 smokers of less than two packs/day, 78 smokers of more than two packs/day) with severe coronary disease. All were initially treated medically and followed up for 15 years. Survival rates for patients with single-, double-, triple-, and left main artery disease were 48, 28, 18, and 9 percent, respectively. Survival rates were 58, 35, 26, and 11 percent, respectively, in 386 patients (118 nonsmokers, 229 smokers of less than two packs/day, 39 smokers of more than two packs/day) considered candidates for bypass surgery. Among the surgery candidates, smoking more than two packs/day was significantly negatively associated with survival when the left main artery cases were excluded. Cardiac survival curves for single-, double-, and triple-artery disease in candidates for surgery, and curves constructed on the basis of 3 percent mortality/artery/year corresponded fairly closely. When an abnormal electrocardiogram (ECG) is considered as a single variable in multivariate analysis, 5-year survival rates of surgical candidates were influenced by (in order of importance) abnormal ECG, symptoms at least 5 years in duration, triple-artery disease, double-artery disease, and arteriosclerosis obliterans. A simple prognostic stratification was devised that used only ECGs and duration of symptoms for each subset based on the number of arteries affected. (Auth. Abs. Mod.)

84–0515. Rogers, R. L.; Meyer, J. S.; Shaw, T. G.; Mortel, K. F.; Hardenberg, J. P.; Zaid, R. R. **Cigarette Smoking Decreases Cerebral Blood Flow Suggesting Increased Risk for**

Stroke. *Journal of the American Medical Association* 250(20): 2796-2800, November 25, 1983.

Decreased cerebral blood flow occurred as a primary effect of chronic smoking in subjects lacking other known risk factors for stroke. The effects of smoking on gray matter blood flow (Fg) were measured using xenon 133 inhalation among 192 subjects without cerebrovascular symptoms: 108 healthy, normal volunteers, and 84 with risk factors for stroke (hypertension, hyperlipidemia, diabetes mellitus, and/or heart disease). Of both risk and nonrisk groups, 75 were habitual smokers (0.5 to 3.5 packs/day for 25 years). Comparisons of mean Fg values for both hemispheres showed significant reductions related to tobacco consumption and risk factors for stroke. Multiple regression equations using smoking, age, risk, and alcohol consumption indicated a combined R^2 value of 0.22. Smoking seems to be a potent risk factor decreasing cerebral blood flow probably by enhancing cerebral arteriosclerosis. Chronic cigarette smoking in persons with other risk factors further reduced Fg values in an additive manner when compared with subjects who had corresponding risk factors but did not smoke. (Auth. Abs. Mod.)

84–0516. Rosenberg, L.; Miller, D. R.; Kaufman, D. W.; Helmrich, S. P.; Van de Carr, S.; Stolley, P. D.; Shapiro, S. **Myocardial Infarction in Women Under 50 Years of Age.** *Journal of the American Medical Association* 250(20): 2801-2806, November 25, 1983.

In a case—control study of 255 women with myocardial infarction (MI) and 802 controls, results showed that the relative risk of MI in women under age 50 increased with the amount smoked. Of all risk factors identified, smoking was the most prominent. Among the MI patients, 9 percent were nonsmokers, 7 percent ex-smokers, 3 percent light smokers, and over 80 percent smoked from 15 to over 35 cigarettes/day. The estimated risk of MI for current smokers of ≥ 35 cigarettes/day was 10 times that of women who never smoked; an estimated 65 percent of MIs were attributable to cigarette smoking. The relative risk of MI increased markedly with increasing levels of total plasma cholesterol and decreasing levels of high-density lipoproteins, and the effects of the two factors appeared to be independent. Other factors significantly associated with MI were hypertension, angina pectoris, diabetes mellitus, blood group A, and a history of MI or stroke before age 60 in a mother or sibling. Factors not significantly associated with MI were obesity, history of preeclamptic toxemia, and type A personality. Women who were postmenopausal appeared to have a lower risk of MI than premenopausal women of similar ages. (Auth. Abs. Mod.)

84–0517. Salonen, J. T.; Puska, P.; Nissinen, A. **Intake of Spirits and Beer and Risk of Myocardial Infarction and Death—A Longitudinal Study in Eastern Finland.** *Journal of Chronic Diseases* 36(7): 533-543, 1983.

The age-adjusted risk of acute myocardial infarction (AMI) among smokers who consumed alcohol less than once/week was over 3 times that of nonsmokers who used alcohol at least once/week. The drinking and smoking habits of 4,063 men (aged 30 to 59) from Eastern Finland were determined by questionnaire in 1972. During 7 years of followup, 209 men experienced an AMI and 223 men died. The intake of beer or of other alcoholic drinks had a strong positive association with smoking and serum triglycerides, a weak positive association with diastolic blood pressure, but no relationship to serum total cholesterol. Use of spirits at least once a week was associated with a reduced risk of AMI. The relative risk adjusted for age and conventional coronary risk factors was 0.5. Consumption of beer had no significant relationship to the risk of AMI. Consumption of at least five bottles of beer/week was related to a slightly excessive risk of death from any cause. Both the use of spirits and smoking were independently related to liver disease risk. (Auth. Abs. Mod.)

84–0518. Seltzer, C. C.; Daly, L. E.; Mulcahy, R.; Graham, I. M.; Hickey, N. **Effect of Stopping Smoking After Unstable Angina and Myocardial Infarction. (Letters).** *British Medical Journal* 287(6401): 1301-1302, October 29, 1983.

A previous article (*British Medical Journal* 287 (6388): 324-326, July 30, 1983) demonstrating the benefits of stopping smoking after myocardial infarction is criticized because nonsmokers showed higher long-term mortality rates than ex-smokers (but lower than smokers). Furthermore, followup began 2 years after the initial attack, yet it is noted that 13 percent (19 of 147) of the nonsmokers compared with only 8 percent (34 of 408) of the smokers died within the original 2-year period. These findings are considered incompatible with the traditional cause—effect hypothesis. The authors reply that the nonsmokers (1) had a higher total burden of risk factors other than smoking; (2) had higher blood pressure, exercised less, and were older than the smokers; and (3) comprised a greater proportion of subjects who had suffered severe or complicated infarction. Thus, many of the smokers would never have had an infarct if they had never smoked. Therefore, cessation of smoking is the best single action in management of coronary heart disease patients. See also 84-0171

84–0519. Simon, J.; Cajzl, L.; Svojgrova, M. **Latent Ischaemic Heart Disease Among the Male Employees of an Industrial Enterprise.** *Cor et Vasa* 25(2): 108-117, 1983.

Smoking showed a correlation with hypercholesterolemia, but not latent ischemic heart disease among 3,758 male industrial workers, aged 40 to 50 years. A total of 291 cases of suspected myocardial ischemia (7.7 percent) were detected by resting ECG, and 311 cases (8.3 percent) of latent ischemic heart disease (IHD) were found using a standard cardiovascular questionnaire; 47 other men were positive both on resting ECG (ischemic changes) and on the questionnaire. Latent IHD was equally frequent among blue and white collar workers. Ischemic ECG findings were positively associated with a positive questionnaire, presence of hypertension, and hypertension or stroke in family history, and inversely with alcohol consumption. Nonsmoking, ex-, and moderate smoking status (<15 cigarettes/day, n=2,461), was significantly associated with increased serum cholesterol ($p<0.001$), but not heavy smoking (n=1,285). No relationship was found between ischemic ECG and smoking, hypercholesterolemia, obesity, education, and physical activity at work or during leisure time. (Auth. Abs. Mod.)

84–0520. Smith, J. W.; Buckels, L. J.; Carlson, K.; Marcus, F. I. **Clinical Characteristics and Results of Noninvasive Tests in 60 Diabetic Patients After Myocardial Infarction.** *American Journal of Medicine* 75: 217-224, August 1983.

Of 861 patients who had survived myocardial infarction, there were fewer smokers among the 60 diabetics (68.3 percent smokers) compared with nondiabetics (84.4 percent smokers). Diabetics had more similarities than differences when compared with nondiabetic patients. Of the primary risk factors, hypertension and mean cholesterol level were significantly higher in diabetics, as was a history of ischemic heart disease. The course in the coronary care unit was similar for both the diabetic and nondiabetic patient groups. There were no striking differences in left ventricular ejection fractions or results of 24-hour Holter monitoring between the groups. A higher percentage of diabetic patients was excluded from taking the limited activity treadmill test, primarily because of angina after infarction, which may account for the observation that the percent of patients with an ischemic response to exercise and the percent with exercise-induced ventricular ectopy did not differ between diabetic and nondiabetic groups. (Auth. Abs. Mod.)

84–0521. Sparrow, D.; Thomas, H. E., Jr.; Weiss, S. T. **Coronary Heart Disease in Police Officers Participating in the Normative Aging Study.** *American Journal of Epidemiology* 118(4): 508-513, October 1983.

Because there is evidence that suggests male police officers are at a higher risk of developing coronary disease than males in the general population, 200 police officers and 1,428 nonpolice officers were followed for 10 years to determine the incidence of disease. Subjects were participants of the Normative Aging Study, a longitudinal study of aging initiated in 1963 at the Veterans Administration Outpatient Clinic in Boston, Massachusetts. Comparison of police officers and nonpolice officers showed no statistically significant differences in the crude incidence rates of coronary heart disease (myocardial infarction, angina pectoris, or death from coronary heart disease) and myocardial infarction alone. When baseline age, serum cholesterol, blood pressure, cigarette smoking, and body mass index were taken into consideration in multiple logistic regression analyses, the lack of significant differences between groups persisted. Police officers relative to nonpolice officers had an adjusted risk ratio (odds ratio) of 1.4 for coronary heart disease and 1.0 for myocardial infarction. Differences between occupational groups were not statistically significant. (Auth. Abs.)

84–0522. Talbott, E.; Kuller, L. H.; Murphy, P. A. **The Epidemiology of Sudden Coronary Death.** *Journal of the South Carolina Medical Association* 79(10): 574-586, October 1983.

Sudden death from coronary heart disease was found to be most strongly related to cigarette smoking in a 24-year followup of the Framingham cohort. Among smokers, the rate of sudden death in men in the youngest decade was almost four times greater than that of nonsmokers; the rate was twice as high in older age groups. In women, no relationship was apparent. In two other studies, smoking was the most important risk factor for sudden coronary death in white women. In another investigation of 163 women (ages 15 to 44) who had fatal heart attacks, only smoking was a significant factor among those who had not suffered previous heart attacks. Other factors for which epidemiological evidence was reviewed were elevated blood pressure, elevated serum cholesterol levels, obesity, diabetes, alcohol use, psychosocial factors, and degree of stenosis.

84–0523. Taylor, C. B.; Houston-Miller, N.; DeBusk, R. F. **Psychosocial Factors: Interventions to Reduce Sudden Death Following an MI.** *Journal of the South Carolina Medical Association* 79(10): 604-610, October 1983.

The most important psychosocial risk factor following myocardial infarct (MI) appears to be the continuation of smoking. Many studies have shown increased risk of another MI or sudden coronary death (SCD) for patients who continue smoking and a reduced risk for those who stop. Depending on the method used, 28 to 72 percent of patients who smoked pre-MI stopped from 6 to 23 months post-MI. Seven possible risk factors for SCD have been identified: (1) sustained disturbing emotions (including anxiety and depression); (2) the Type A behavior pattern; (3) work-related overload; (4) socioeconomic disadvantage; (5) lifestyle-related risks; (6) acute stressful or emotional situations; and (7) mitigating circumstances, such as effective social support, the seriousness of existing disease, and medical treatment. These factors can all be accurately assessed with psychological tests, but some (e.g., overload and social support) are best evaluated through direct questioning. The need for the designing and testing of intervention methods is described.

84–0524. Watts, E. J.; Lewis, S. M. **Spurious Polycythaemia—A Study of 35 Patients.** *Scandinavian Journal of Haematology* 31(3): 241-247, September 1983.

Results from 35 patients with elevated hemoglobin and hematocrit showed that smoking and obesity in such patients are sometimes associated but do not progress to polycythemia vera. Patients had normal or borderline red cell mass, and it was confirmed that the most common disease association was hypertension. Also, the peripheral blood findings are reversible when blood pressure is treated, especially when the body hematocrit is low. In patients who smoked more than 20 cigarettes/day, average red cell mass was 111 percent. It is concluded that spurious polycythemia frequently coexists with smoking, obesity, and peripheral vascular disease; that the excess morbidity of the condition is determined by the underlying disease; and that it is more rational to treat this than to attempt to alter the blood volume by venesection. (Auth. Abs. Mod.)

84–0525. Webber, L. S.; Hunter, S. M.; Baugh, J. G.; Srinivasan, S. R.; Sklov, M. C.; Berenson, G. S. **The Interaction of Cigarette Smoking, Oral Contraceptive Use, and Cardiovascular Risk Factor Variables in Children: The Bogalusa Heart Study.** *American Journal of Public Health* 72(3): 266-274, March 1982.

Results suggest that the lipid and lipoprotein response to cigarette smoking and oral contraceptive (OC) use increases cardiovascular risk. Two separate surveys were conducted (from 1976 to 1977 and 1978 to 1979) in a total biracial population of children (aged 8 to 17 years) to determine the effects of cigarette smoking and OCs on blood pressure and serum lipids and lipoproteins. Information regarding smoking habits and OC use was obtained from detailed questionnaires. Since current smoking was most prevalent among 14- to 17-year-olds, examinations were limited to that age group: 1,281 in 1976 to 1977 (95 percent supplying information on smoking behavior); and 953 in 1978 to 1979 (99 percent supplying information on smoking behavior). For white boys and white and black girls, a small but statistically significant decrease in diastolic blood pressure levels for cigarette smokers when compared to nonsmokers was noted. In both surveys, significant increase among cigarette smokers in β-lipoprotein cholesterol, pre-β-lipoprotein cholesterol, and triglycerides as well as a decrease in α-lipoprotein cholesterol was noted, especially for white girls. OC users had higher total cholesterol and β-lipoprotein cholesterol and lower α-lipoprotein cholesterol than nonusers. After adjusting for OC use, particularly among white girls, cigarette smokers still demonstrated high pre-β-lipoprotein cholesterol and lower α-lipoprotein cholesterol levels than nonsmokers. (Auth. Abs. Mod.)

84–0526. Zalokar, J. B. **Leukocyte Counts and Coronary Heart Disease in a Japanese Cohort. (Letter).** *American Journal of Epidemiology* 118(4): 611-612, October 1983.

Data on coronary heart disease (CHD) incidence in Western populations are compared with an earlier report (*American Journal of Epidemiology* 116(3): 496-509, September 1982) on rates in a Japanese cohort, in terms of smoking habits, leukocyte counts, and cholesterol levels. While the Japanese study did not analyze the relationship between smoking and leukocytes for lack of information, in the Western studies, myocardial infarction (MI) among current smokers was related to elevated leukocyte counts. This finding was not true for MI patients who were nonsmokers. It is suggested that the leukocyte count adds information either about the quantity of inhaled smoke or about individual sensitivity to cigarette smoke with consequently increased risk of MI. Smoking was a weak risk factor for CHD in the Japanese cohort (stronger for MI than for angina pectoris), suggesting that leukocyte count information on quantity of smoke inhaled would not be relevant to CHD risk in that population. It is also noted that the difference in fat consumption (16 percent of total calories in Japan compared with about 40 percent in the United States and France) may account for much of the differences in CHD risk factors found in these populations. See also 83-0145.

See also, **84–0419, 84–0479, 84–0658**

PREGNANCY AND INFANT HEALTH

84–0527. Curet, L. B.; Rao, A. V.; Zachman, R. D.; Morrison, J.; Burkett, G.; Poole, W. K. **Maternal Smoking and Respiratory Distress Syndrome.** *American Journal of Obstetrics and Gynecology* 147(4): 446-450, October 15, 1983.

The infants of smoking mothers showed both lower birth weights and reduced incidence of respiratory distress syndrome. Infants of 603 patients, for whom information about smoking habits during pregnancy was available, were studied for incidence of respiratory distress syndrome. Among the 360 patients who did not smoke, the incidence of respiratory distress syndrome in the neonate was 15.1 percent, whereas among smoking patients ($n=360$, $198<1$ pack/day, $45 \geq 1$ pack/day), the incidence was 9.1 percent. It is speculated that maternal smoking produces a condition of chronic stress in the fetus that brings about an acceleration of fetal pulmonary maturation. (Auth. Abs. Mod.)

84–0528. Golding, J.; Butler, N. R. **Maternal Smoking and Anencephaly.** *British Medical Journal* 287(6391): 533-534, August 20, 1983.

Although available evidence suggests that maternal smoking is associated with reduced birth weight and perinatal loss, results indicate that it is not important in the etiology of anencephaly. Data on 491 pregnancies from the 1958 British Mortality Survey were compared with singleton deliveries for anencephalic incidence, maternal smoking history, and social class. Incidence/1,000 births if the mother smoked at start of pregnancy was 2.89, significantly greater than for nonsmokers (2.18). However, incidence of anencephaly rose sharply as social class fell, from 1.42 in classes I and II, to 3.16 in classes IV and V. Indirect standardization for each of the five social classes resulted in no significant differences between the observed and expected numbers in each smoking group. Findings suggest that lack of vitamin C in smokers is not a factor in development of anencephaly.

84–0529. Harrison, G. G.; Branson, R. S.; Vaucher, Y. E. **Association of Maternal Smoking With Body Composition of the Newborn.** *American Journal of Clinical Nutrition* 38(5): 757-762, November 1983.

Results suggest that the reduction in birth weight in newborns of mothers who smoked during pregnancy is due primarily to reduction in lean body mass, while deposition of subcutaneous fat is relatively unaffected. The relationship of maternal cigarette smoking to anthropometric measurements of the newborn was investigated in 285 full-term Caucasian infants. Mothers were classified as smokers, if they reported smoking both before and during pregnancy ($n=109$), and nonsmokers ($n=176$). Infants of smoking mothers were significantly lighter, shorter, and had smaller head and arm circumferences than those of nonsmokers, consistent with previous findings. Ponderal index was not different in the two groups, and there were no differences in any of the skinfold measurements or in calculated cross-sectional fat area of the upper arm. Multiple regression analyses revealed independent effects of smoking on weight, length, and arm circumference, but no independent effect of smoking on any index of subcutaneous fat. This differential effect on lean vs. adipose tissue deposition differs from that expected if the effect of smoking on fetal growth is mediated by energy availability. (Auth. Abs. Mod.)

84–0530. Heikkinen, R.; Lehtovirta, P. **Aidin Tupakointi on Sikion Ongelma. [Smoking by the Mother Is a Problem for the Fetus.]** *Duodecim* 99(14): 856-858, 1983, Finnish.

Smoking during pregnancy has been shown to slow fetal growth, although the pathogenesis has not been established. The elevated maternal carboxyhemoglobin levels that result from smoking make oxygen less available to the fetus while lowering the partial pressure of the fetal arterial blood. Carbon monoxide diffuses slowly through the placenta. Elevation of fetal carbon monoxide levels is often found several hours after smoking by the mother, and remains elevated longer in the fetal than in the maternal circulation, leading to hypoxia. Nicotine causes maternal catecholamine release, resulting in a contraction of the uterine arteries and a decrease in placental blood flow, which also leads to hypoxia. Nicotine crosses the placenta, often reaching higher fetal than maternal levels and causing fetal adrenergic activation. Fetal defense against maternal smoking occurs via catecholamine release, primarily noradrenalin, which increases blood flow and helps protect the oxygen supply. It is noted that the fetal heart works longer under hypoxic conditions, and persistence of this hypermetabolic state may be responsible for the decreased fetal growth.

84–0531. Lampe, L.; Batar, I.; Bernard, R. P. **Effects of Hypertensive Disorders on the Outcome of Pregnancy: Early Findings of Maternity Care Monitoring (MCM) in Debrecen, Hungary.** *Clinical and Experimental Hypertension* B1(1): 1-37, 1982.

An analysis of 8,818 singleton births, documented in the maternity care monitoring (MCM) system, revealed that smoking has a more harmful effect on pregnancy outcome than does hypertension. The relationship between pregnancy outcome and maternal hypertensive disorders in pregnancy (HDP) was examined with respect to occurrence, primary complications of labor and delivery, fetal distress, antenatal fetal loss, prenatal visits, and smoking. Maternal smoking increased risk of stillbirth by 278 percent (HDP, 211 percent), decreased birth weight 282.6 g (HDP, 18.6 g), and was associated with a 68.7 percent increase in preterm births (HDP, 35.0 percent). Also, smokers tend to be at an increased risk from inadequate prenatal visit frequency. An overview of the startup and organization of the MCM testing site at Debrecen is provided.

84–0532. Newcombe, R. G. **Reversal of Changes in Distribution of Gestational Age and Birth Weight Among Firstborn Infants of Cardiff Residents.** *British Medical Journal* 287(6399): 1095-1096, October 15, 1983.

The proportion of primiparous women who had never smoked increased from 56.0 percent during the 1970 to 1974 period to 61.3 percent between 1975 and 1979. Firstborn singleton infants born to Cardiff residents during the decade 1970 to 1979 were studied. Both mean birth weight and gestational age at delivery rose during the period. Changes in maternal age, height, smoking habits, and history of spontaneous or induced abortion did not explain these findings. It is noted that women in whom birth was induced were less often smokers, unmarried, or of short stature, and were more often of high social class and over 20 years old. The decreased proportion of infants weighing less than 2,500 g may be explained by the overall increase in gestational age, which in turn may have resulted from decreased use of induction of labor during 1975 through 1979. (Auth. Abs. Mod.)

84–0533. Olsen, J.; Rachootin, P.; Schiodt, A. V. **Alcohol Use, Conception Time, and Birth Weight.** *Journal of Epidemiology and Community Health* 37(1): 63-65, March 1983.

Low birth weight was significantly related to tobacco use in the year of delivery ($p<0.01$), alcohol use during the same period ($p<0.05$), and a delay in conception of over 6 months ($p<0.01$). Predictors of birth weight and birth length were studied using sociodemographic data collected from 2,259 women who resided in Funen County, Denmark, and who had delivered a healthy child during the 1978 to 1979 period at Odense University Hospital. Subjects were classified either as smokers or nonsmokers, and assigned to one of four categories according to level of alcohol consumption. Other independent variables included oral contraceptive use, age, parity, and education. Smoking history and a delay in conception were also found to be significantly associated with short birth length ($p < 0.01$ and $p < 0.01$, respectively). (Auth. Abs. Mod.)

84–0534. Rantakallio, P. **A Follow-Up Study Up to the Age of 14 of Children Whose Mothers Smoked During Pregnancy.** *Acta Paediatrica Scandinavica* 72(5): 747-753, September 1983.

The children of mothers who smoked during pregnancy (n=1,819), all born in northern Finland in 1966 and followed up to the age of 14, were more prone to respiratory diseases than children of nonsmoking controls. They were also shorter and showed less mean ability at school. The differences remained significant after adjusting for the mother's height, age, and social class (determined by father's occupation), number of older and younger children in the family, and child's sex. The smoking mothers were found to differ from controls in each social class (in contrast to the situation at the time of pregnancy), and were currently less healthy, more often unemployed, and had more often left their families. Maternal smoking had an effect on children's physical and mental development when these factors were taken into consideration in regression analyses, although maternal smoking was less important than many other sociobiological factors, and no more important than paternal smoking. (Auth. Abs. Mod.)

84–0535. Verma, R. C.; Chansoriya, M.; Kaul, K. K. **Effect of Tobacco Chewing by Mothers on Fetal Outcome.** *Indian Pediatrics* 20(2): 105-111, February 1983.

Significantly lower birth weight (average 395.3 g less) and birth length (0.518 cm less) were observed among infants of Indian mothers who chewed or ate tobacco during pregnancy (n=70), compared with infants of nonconsumers (n=70). Tobacco users and controls were precisely matched for socioeconomic status; maternal literacy, parity, age, height, and weight; birth interval; and infants' gestational age and sex. Infants' birth weight, birth length, and skull circumference were studied. Birth weight was particularly affected when maternal tobacco consumption exceeded 200 mg/day, in forms more likely to be ingested than chewed (Pan, Zarda, and Biri Patti).

OTHER DISEASES AND CONDITIONS

84–0536. De Vernejoul, M. C.; Bielakoff, J.; Herve, M.; Gueris, J.; Hott, M.; Modrowski, D.; Kuntz, D.; Miravet, L.; Ryckewaert, A. **Evidence for Defective Osteoblastic Function. A Role for Alcohol and Tobacco Consumption in Osteoporosis in Middle-Aged Men.** *Clinical Orthopaedics and Related Research* 179: 107-115, October 1983.

In a group of 11 men (aged 35 to 50 years) with idiopathic osteoporosis, 8 were mild alcoholics and 10 were heavy smokers (over a pack/day for at least 20 years). Two had absorptive hypercalciuria. Histomorphometry showed that the patients had low trabecular bone volume and mean trabecular thickness when compared with age-matched control subjects. Mean wall thickness was also markedly reduced in patients compared with control subjects. The quantity of resorbed bone was extrapolated from the calculated mean interstitial bone thickness. Resorption was not significantly different in patients and control subjects. Consequently, in this group of patients with severe osteoporosis, the pathogenesis was characterized by markedly decreased bone formation. While no clinical controlled study of decreased axial bone mass in male smokers and nonsmokers has been performed, smoking seems to be a predisposing factor for postmenopausal osteoporosis, and cannot be excluded as a possible risk factor for osteoporosis in men. (Auth. Abs. Mod.)

84–0537. Faglia, G.; Arosio, M. **Fumo e Sistema Endocrino. [Smoking and the Endocrine System.]** *Minerva Medica* 73(36): 2337-2344, September 22, 1982, Italian.

The toxicity of cigarette smoke on the endrocrine system, which glands are affected, and the contribution of these endocrine modifications to smoking-related morbidity are reviewed. The influence of nicotine on vasopressin, corticotropin, and the somatotropic, thyrotropic, and gonadotropic hormones is discussed. Smoking's primary effect is a nicotine-provoked release of catecholamines, which contributes to the vascular damage associated with cigarette use. Smoking also results in earlier onset of menopause, more acute pre-senile osteoporosis, and a possible increase in secondary amenorrhea.

84–0538. Gooch, P. **Tobacco and Health: Scientists Investigate the Relationship.** *Tobacco Reporter* 110(10): 36-38, 40, October 1983.

Although previously dominated by the tobacco industry, a recent change in direction and funding procedures has given the Tobacco and Health Reserch Institute freedom to investigate any relevant health interest without fear of results. The Institute, in Lexington, Kentucky, is directed to "prove or disprove the charges against tobacco products and to preserve and strengthen the tobacco industry and tobacco programs in Kentucky." Ongoing research, divided into five categories (pulmonary, cardiovascular, neurobehavioral, passive smoking/smoke components, plant modification), is oriented toward minimizing smoking-related health risks, identifying smokers who are at high risk, and determining why people smoke. Many current projects utilize an improved mainstream—sidestream peristaltic pump smoke exposure system that individually measures separate cigarette smoke components and precisely controls animal dosage. A microcomputer calculates individual smoking characteristics, enabling testing of larger populations and allowing researchers to expose animals to the same amounts of smoke inhaled by active and passive smokers. Animal studies examine physiologic, toxicologic, and pathologic smoke inhalation effects. Current studies include nicotine binding sites in the brain, cigarette smoke effects on pulmonary macrophages, nicotine aerosol and cigarette smoke effects on breathing, and cardiovascular disease relationship with cigarette smoking. Atherosclerosis development is discussed in terms of prostacyclin—thromboxane imbalance.

84–0539. Hirsch, J.-M.; Johansson, S. L. **Effect of Long-Term Application of Snuff on the Oral Mucosa: An Experimental Study in the Rat.** *Journal of Oral Pathology* 12(3): 187-198, June 1983.

Exposure of rats to snuff for 10 to 16 hours/day, 5 days/week, for most of their lifespan resulted in lesions mainly restricted to the epithelium and the underlying connective tissue of the test canal. The long-term effect of snuff exposure was studied in Sprague-Dawley rats using a surgically created test canal in the lower lip to retain snuff. The rats received standard snuff (n=42) and highly alkaline snuff (n=10) for 9 to 22 months. Untreated rats with identical test canals (n=15) served as controls. Complete post mortem examinations were performed. One rat exposed to standard snuff for 9 months developed a squamous cell carcinoma of the oral cavity. However, exposure to standard snuff usually resulted in a mild to moderate hyperplasia of the epithelium, hyperorthokeratosis, and acanthosis. Rats exposed to snuff for 18 to 22 months showed vacuolated cells penetrating deeper into the epithelium with hyperplastic and atrophic lesions. In a few rats, severe dysplastic changes developed in the crevicular epithelium. Rats exposed to alkaline snuff differed little from the first group except that there was focally atrophic and ulcerated epithelium and less fibrosis. Pathological findings outside the oral cavity were rare. Squamous cell hyperplasia of the forestomach was found in rats exposed to snuff for 18 to 22 months, possibly caused by ingested snuff. (Auth. Abs. Mod.)

84–0540. Jonderko, G.; Konca, A.; Kwiatek, R.; Marcisz, C.; Neumann, M. **Hypersensitivity to Tobacco Antigens in Smokers and Nonsmokers.** *Allergie und Immunologie* 28(3): 187-191, 1982.

No evidence was found for an essential reagin-type allergic reaction to tobacco smoke among 336 current smokers, 91 ex-smokers, 175 subjects indirectly exposed to smoke, and 112 non-smoke-exposed subjects. All were administered a dermic allergometric test with tobacco extract and examined for atopic bronchial asthma and urticaria. The subjects indirectly exposed to tobacco showed the highest frequency of reactions to tobacco allergens. No significant relationship was found between incidence of asthma and urticaria according to tobacco smoke exposure. However, chronic bronchitis was observed in 40.5 percent of current smokers and in 50.0 percent of ex-smokers.

84–0541. Luzzatto, R.; Maciel, S. R. **Curschmann Spirals in the Vagina. (Letter).** *Annals of Internal Medicine* 99(2): 278-279, August 1983.

Among eight cases of Curschmann spirals found in 9,474 consecutive cervical smears, only two were moderate smokers and the remaining six were nonsmokers. A letter published in 1974 suggested a relationship between the spirals and the systemic effects of cigarette smoking. It is noted, however, that all eight of the subjects had a history of food allergy or contact dermatitis, suggesting an immunoallergic rather than smoking-related cause for the spirals.

84–0542. Mackay, J. M.; Fox, D. P.; Brunt, P. W.; Johnston, A. W. **Increased Sister-Chromatid Exchange Frequency in Patients Receiving Sulphasalazine Therapy for Ulcerative Colitis.** *Mutation Research* 118(3): 205-212, August 1983.

Sister-chromatid exchange (SCE) and micronucleus frequencies are reported for lymphocytes cultured in vitro from 15 patients receiving sulphasalazine therapy for ulcerative colitis and 15 controls matched for age and sex. Cigarette smoking is an important inducer of SCEs, but there was no systematic difference between the two groups in smoking (or drinking) habits. While the patients did not differ from matched controls for micronucleus frequency, there was a substantial rise in their SCE frequency. This evidence of DNA damage is discussed in relation to the known cancer risk that ulcerative colitis carries, and the possibility of an increased mutation rate in the germ cells of those receiving sulphasalazine therapy. (Auth. Abs. Mod.)

84–0543. Penny, W. J.; Penny, E.; Mayberry, J. F.; Rhodes, J. **Mormons, Smoking, and Ulcerative Colitis. (Letter).** *Lancet* 2(8362): 1315, December 3, 1983.

Smokers appear to be at three times the risk of ulcerative colitis, compared with nonsmokers; however, ulcerative colitis is about four times more common in Mormons, who largely abstain from smoking. A survey of 57,000 of 100,000 Mormons residing in Great Britain, involving 230 of a possible 342 Mormon church units, indicates a five times greater incidence of ulcerative colitis than of Crohn's disease. Previous determinations have shown ulcerative colitis to be about twice as common as Crohn's disease. Crohn's disease was found in a proportion similar to that seen in other Britons among active church members surveyed (n=17,700). However, ulcerative colitis prevalence was five times greater than it was in an Oxford study completed 20 years ago, and three times greater than in Copenhagen, which has the highest reported prevalence of the disease (117 cases/100,000 population).

84-0544. Schmidt, F. **Passivrauchen—Nur Eine Belastigung Oder Echtes Gesundheitsrisiko? [Passive Smoking—Only an Inconvenience or a Health Risk?]** *Oeffentliche Gesundheitswesen* 45(5): 249-251, May 1983, German.

Criticism of passive smoking research (*Deffentliche Gesundheitswesen* 44: 454-456, 1982) is refuted. The earlier article claimed that (1) genuine passive smoking research with animals has never been performed; (2) the dilution factor makes passive smoking risks irrelevant; and (3) the results of epidemiological studies are overrated. In response, it is stated that (1) all animal smoking experiments are actually passive smoking studies because laboratory animals cannot be made to inhale as human smokers do; (2) although dilution means the passive smoker is exposed to only one-half cigarette/day, the sidestream smoke content of nitrosamines is 50 times higher than that of mainstream smoke, rendering the dilution factor unimportant; and (3) even if only a few studies found a significant increase in lung cancer and respiratory illness among people who live with smokers, the tendency should be considered important. See also 83-1727.

84-0545. Steinfeld, J. L. **Women and Children Last? Attitudes Toward Cigarette Smoking and Nonsmokers' Rights, 1971.** *New York State Journal of Medicine* 83(13): 1257-1258, December 1983.

Surgeon General Jesse L. Steinfeld's semiofficial call for a nonsmokers' rights movement at the 1971 meeting of the Interagency Council on Smoking and Health has been followed over the past 12 years by a number of city, state, and Federal regulations protecting the nonsmoker. Excerpts of Dr. Steinfeld's address, which was promptly and severely criticized by others at the Department of Health, Education, and Welfare, are provided on three major issues: (1) effects of smoking during pregnancy; (2) women and smoking (with special concerns regarding nurses, advertising, and anticipated mortality trends); and (3) nonsmokers' rights to nonpolluted air. Dr. Steinfeld indicated that continued warnings of smoking-related health hazards would be less effective than social actions such as peer and other pressures, and legislation to protect the nonsmoker. A subsequent Surgeon General's report summarizing all available data on the effects of passive smoking was followed by the first requirement by the General Services Administration for the protection of nonsmokers in Government office buildings.

84-0546. Weiss, S. T.; Tager, I. B.; Schenker, M.; Speizer, F. E. **The Health Effects of Involuntary Smoking.** *American Review of Respiratory Disease* 128(5): 933-942, November 1983.

A relatively large body of data relates parental (particularly maternal) cigarette smoking to the occurrence of both acute respiratory illnesses and chronic respiratory symptoms in children. The effect seems to be greatest early in life and cannot be separated from in utero exposure. While data linking parental smoking to lower levels of pulmonary function are all cross-sectional and less conclusive, the magnitude of the direct effect of passive smoke exposure is likely to be relatively small (from 1 to 5 percent reduction in maximally obtained lung function level in exposed children). The important effects of passive smoke exposure (defined as the exposure of nonsmokers to tobacco combustion products in an indoor environment) in childhood are twofold: the slight reduction in pulmonary function level may predispose an individual to increased risks from environmental agents later in life; and having a parent who smokes substantially increases the likelihood that a child will become a smoker. Involuntary smokers are exposed to a quantitatively smaller and qualitatively different smoke exposure than active smokers. Quantitation of exposure is particularly difficult in both physiologic and epidemiologic studies. Acute physiologic studies have documented minimal physiologic changes in healthy subjects. However, individuals with heart or lung disease may be differentially affected. Data on adults are insufficient to allow a quantitative estimate of effects, but two studies have linked lung cancer in nonsmokers to exposure to spouses' cigarette smoke. Further research is needed to confirm findings on passive smoking. (Auth. Abs. Mod.)

84-0547. Wickre, C. G.; Norman, D. J.; Bennison, A.; Barry, J. M.; Bennett, W. M. **Postrenal Transplant Erythrocytosis: A Review of 53 Patients.** *Kidney International* 23(5): 731-737, May 1983.

Smoking, diabetes, and a rejection-free course of treatment were found to be risk factors for the development of posttreatment erythrocytosis in a review of 237 renal transplant patients. There was a 17.3 percent (n=53) incidence of posttransplant erythrocytosis. The influence of kidney source, pretransplant hematocrit, duration of pretransplant dialysis, renal transplant function, acute rejection, transplant renal artery stenosis, urinary tract obstruction, smoking, diabetes, retention of native kidneys, splenectomy, parathyroidectomy, immunosuppression, hypertension, and liver enzyme abnormalities on the development of erythrocytosis in 53 recipients was determined. A total of 59.6 percent had a history of smoking compared with 34.7 percent of controls. In contradistinction to previous smaller series, erythrocytosis occurred in patients with good renal function (serum creatinine 1.62±0.43 mg/dl) without prominence of graft rejection, transplant artery stenosis, or obstruction. Despite therapeutic phlebotomy, 11 thromboembolic events occurred in 10 of the 53 patients with erythrocytosis, but in none of the controls ($p<0.001$). (Auth. Abs. Mod.)

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

84–0548. Ashton, H. **Patterns of Smoking. Social and Psychological Factors.** *Practitioner* 227(1383): 1415-1423, September 1983.

An overview of the psychological, social, and cultural influences that support the smoking habit is presented. The onset of cigarette use is the least understood aspect of the habit, and is discussed with respect to the role of friends, family members, and cultural and personality factors. Nicotine as the motive for continued smoking and the development of nicotine dependence are also reviewed. Issues and methods in giving up smoking, including trends in smoking prevalence and means of primary prevention, are covered. Smoking on a worldwide scale and trends in developing countries are explored.

84–0549. Biglan, A.; Severson, H.; Bavry, J.; McConnell, S. **Social Influence and Adolescent Smoking: A First Look Behind the Barn.** *Health Education* pp. 14-18, September-October 1983.

Questionnaire data on smoking history, current smoking practices of subjects, family members, and friends, and attitudes toward smoking were collected from 518 teenagers in grades 6 to 8 and 10 to 12. Breath samples were analyzed for carbon monoxide levels. Most adolescents (70 percent) reported getting their first cigarette from another person (57 percent from a friend), and most (73 percent) were with another teenager when they first smoked. A Guttman scale of transition to smoking revealed a reproducibility of 0.99 (minimum marginal reproducibility of 0.81), indicating that an accurate prediction of response can be made. A 1-week self-monitoring study was done on 29 adolescent smokers to reveal the specific situational variables that affect the initiation and development of smoking. Greater compliance was achieved with those subjects who received daily telephone calls from study personnel. Results will be used to help in the development of programs to deter adolescents from beginning to smoke.

84–0550. Botvin, E. M.; Botvin, G. J.; Baker, E. **Developmental Changes in Attitudes Toward Cigarette Smokers During Early Adolescence.** *Psychological Reports* 53(2): 547-553, October 1983.

Significant differences exist between sixth, seventh, and eighth graders in their attitudes toward the social image of smoking, with sixth graders having the least positive attitudes. The perceptions of 104 sixth, 137 seventh, and 110 eighth graders were examined to identify potential developmental differences. A significant interaction of sex with grade was also evident; the sixth grade boys had the least favorable attitudes toward smokers. No significant differences were found between students who had friends who smoked and those who did not. These findings suggest that, in addition to the many other developmental changes occurring during early adolescence, there is a shift toward a more positive social image of cigarette smoking that is unrelated to the smoking status of friends. (Auth. Abs. Mod.)

84–0551. Boulton, M. G.; Williams, A. **Health Education in the General Practice Consultation: Doctors' Advice on Diet, Alcohol and Smoking.** *Health Education Journal* 42(2): 57-63, 1983.

Analysis of 1,470 tape-recorded consultations from 16 doctors showed that only a small fraction of the opportunities available for giving advice about smoking were used and on no occasion was advice supplemented by written material. Two types of opportunities for giving advice on smoking, diet, and alcohol were identified: (1) problem-related health education, and (2) non-problem-related education, where the doctor could take the initiative in raising the topic. In an intensive sample (405 cases that eliminated consultations in which no new educational issues were raised), there were 105 opportunities for discussing smoking-related problems, but in only 17 percent did the doctor exploit the opportunity. In a random sample of all consultations (n=75), only 10 percent used the opportunities available to discuss smoking habits with their patients. Virtually no attempts were made to explore patient rationale for needing to smoke, and in only one consultation did the doctor justify his view on smoking by explaining its potential effects on the patient. While other studies have indicated that general practitioners accept that they have a significant part to play in helping patients curb smoking habits, these results underline the gap between positive attitudes and actual behavior. Similar results for advice on diet and alcohol are also provided. Time is certainly a major constraint, but limited awareness of available opportunities and lack of skills to take advantage of them also contribute to the low level of activity observed. Attempts to encourage health education in the general practice consultation must deal with doctors who define their role primarily in terms of being responsive to patient demands or needs, and those who view such educational attempts as intrusive and moralizing.

84–0552. Brook, J. S.; Whiteman, M.; Gordon, A. S.; Brook, D. W. **Fathers and Sons: Their Relationship and Personality Characteristics Associated With the Son's Smoking Behavior.** *Journal of Genetic Psychology* 142(2): 271-281, 1983.

Interrelationships between the domains of father personality attributes, father socialization techniques, adolescent personality attributes, and the son's use of tobacco were examined. A sample of 246 male adolescents and their fathers from intact homes was administered questionnaires. Each of the three domains was significantly associated with the son's tobacco use, despite control on the remaining domains. Both father domains and the adolescent personality domain seem to have independent lines of relationships and constitute their own sets of sufficient conditions for adolescent tobacco use. Thus, adverse paternal personality attributes are operative even in the context of effective paternal socialization techniques and positive adolescent personality attributes. Similarly, less effective paternal socialization techniques are associated with tobacco use despite benign paternal and adolescent personality characteristics. Finally, adverse adolescent personality attributes are associated with tobacco use despite optimal paternal conditions. (Auth. Abs. Mod.)

84–0553. Calnan, M. W.; Johnson, B. M. **Influencing Health Behaviour: How Significant Is the General Practitioner?** *Health Education Journal* 42(2): 39-44, 1983.

In a review of the role of the general practitioner as a health educator, it is noted that while there is some evidence that advice is effective in changing people's smoking habits, it is still not clear which aspect of the consultation process makes the crucial impact. The practitioner may provide a cue for the smoker to initially quit, but studies indicate that further support is needed to achieve and maintain abstinence. Features of the general practitioner setting that make it so amenable to the practice of health education are examined, and evidence from studies that have attempted to evaluate the effectiveness of health education provided by general practitioners is reviewed. The compatibility of the doctor—patient relationship in present-day practice with what is required for effective health education is also discussed. It is concluded that there is still a need for systematic evaluative research before decisions are made about whether the busy practitioner should be encouraged to carry out more health education and the most effective form and method that should be used. Also, the relative efficacy of other health care workers as health educators should be investigated.

84–0554. Chait, L. D.; Griffiths, R. R. **Effects of Caffeine on Cigarette Smoking and Subjective Response.** *Clinical Pharmacology and Therapeutics* 34(5): 612-622, November 1983.

A naturalistic laboratory investigation of six smokers showed that caffeine and *d*-amphetamine have different effects on cigarette smoking behavior and subjective response, suggesting that the positive correlation between cigarette smoking and coffee drinking is not the result of a simple pharmacologic effect of caffeine. A within-subject, repeated-measures design was used, and each subject received placebo, caffeine base (50 to 800 mg), or *d*-amphetamine sulfate (25 mg) on several occasions before 90-min daily smoking sessions. There was no evidence of an increase in the number of cigarettes smoked or the amount of smoke inhaled/session after caffeine. Caffeine increased salivary caffeine concentrations, arm tremor, and self-reported measures of mood and subjective response. The major subjective effects of caffeine were increases in tension—anxiety and dysphoric—somatic effects. In contrast, *d*-amphetamine induced increases in the number of cigarettes smoked and in the amount of smoke inhaled/session. The major subjective effects of 25 mg of *d*-amphetamine were increases in measures of well-being, euphoria, and mental efficiency. (Auth. Abs. Mod.)

84–0555. Corty, E. **The Test-Retest Reliability of Measures Used in Cigarette Smoking Research.** *Addictive Behaviors* 8(3): 315-317, 1983.

None of the three measures used in cigarette smoking research was considered adequately reliable following a 2-week assessment period of 49 subjects (mean age 19.1 years) who had smoked an average of 14.8 cigarettes/day for 3.8 years. The 19 males and 30 females filled out questionnaires and were told to return in 2 weeks, at which time they completed the same questionnaires. Given a characteristic that is assumed to be stable and that has been measured with the same instrument at two points briefly separated in time, one measurement would be expected to be highly predictable from the other. That is, the measurement at time 1 (T1) should account for most of the variance in the measurement at time 2 (T2) (the definition of test—retest reliability). In this study, a measure was considered as stable over time if it explained at least 50 percent of the variance in itself 2 weeks later. Results from the Best and Hakstian Survey indicated moderate to high correlations of factors, but when subjects' factor scores were transformed into T scores, the correlations for the males ranged from -0.27 to 0.89; for females, from -0.02 to 0.98. Thus the survey does not demonstrate adequate test—retest reliability. The two other measures investigated, the Condiotte and Lichtenstein Self-Efficacy Scale, and the Epstein and Collins Inventory of Smoking Situations, also produced inadequate test—retest results. Caution is advised when using any of these measures.

84–0556. Fawzy, F. I.; Coombs, R. H.; Gerber, B. **Generational Continuity in the Use of Substances: The Impact of Parental Substance Use on Adolescent Substance Use.** *Addictive Behaviors* 8: 109-114, 1983.

The use of cigarettes, alcohol, marihuana, and hashish by children is strongly related to parental use. Results of interviews with 256 adolescents (aged 13 to 17 years) indicated that when mothers and fathers reported smoking more than one pack of cigarettes/day, 85 and 64 percent of children reported substance use. Children were similar in age, sex, ethnicity, and socioeconomic status. Decreased incidence of substance use was reported by children whose parents smoked less than one pack of cigarettes/day. Higher substance use was reported by children whose parents had quit smoking. Thus, the implications for preventing problems related to substance use are clear: (1) parental abstinence from substances may be the first line of defense against adolescent initiation (social learning explanation); and (2) parental efforts to maintain a quality relationship with their children may reduce emotional problems that could lead to deeper involvement with substances (the family pathology explanation). (Auth. Abs. Mod.)

84-0557. Frederiksen, L. W. **Evaluation of Smoking Risk: Some Proposed Minimum Standards.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 90-95.

The special problems of assessing the risk of smoking in an occupational setting are discussed, noting that while risk is the major concern, an adequate description of smoking involves assessment of what is smoked, the rate of consumption, and smoking topography. Methods used must be held constant across experimental conditions if replicable and reliable results are to be obtained. Also, there should be some analysis of measurement reliability at each assessment point. The advent of biochemical measures has simplified such validation. Requirements are defined for (1) identifying nonsmokers; (2) establishing the point of abstinence for each individual; and (3) setting a minimum period of time for total abstinence. Data for current smokers should not be presented as an index that obscures the independent determination of the smoking rate and substance used. While the suggested criteria do not delineate categories of smokers or specific measures to use, they emphasize (1) the quality of the data, and (2) the detail of the information needed if clear and comparable measures of smoking are to evolve.

84-0558. Galizio, M.; Rosenthal, D.; Stein, F. A. **Sensation Seeking, Reinforcement, and Student Drug Use.** *Addictive Behaviors* 8(3): 243-252, 1983.

Tobacco was the third most frequently used drug (after alcohol and marihuana) among college student users of several drugs (polydrug group) and those among whom marihuana was the only illicit drug used. Nonusers of illicit drugs smoked and used alcohol significantly less than the polydrug and the marihuana groups; the marihuana group used less (but not significantly less) tobacco and alcohol than the polydrug group. Members of all three groups completed the Sensation Seeking Scale, a drug use survey, and provided demographic information. Subjects were also asked to generate a list of the events that they found most reinforcing (Reinforcer List). Drug use was positively related to scores on all subscales of the Sensation Seeking Scale. Although neither drug use nor sensation seeking scores were related to the total number on items generated on the Reinforcer List, drug users did generate a higher percentage of items rated as high sensation producing items, and percent high sensation reinforcers was correlated with scores on the Sensation Seeking Scale. Results illustrate the importance of the sensation seeking motive as a correlate of student drug use. (Auth. Abs. Mod.)

84-0559. Gordon, N. P. **De-Fusing the Symbolic Value of Smoking. (Letter).** *American Journal of Public Health* 73(9): 1102, September 1983.

A rebuttal of a previous article's premise (*American Journal of Public Health* 73(4): 439-441, April 1983), which stated smoking serves primarily to make some adolescents feel grown up, charges that antismoking programs targeted solely at students and not the school society as a whole will only reinforce smoking as a symbol of adulthood. The earlier study addressed smoking's relationship to social status configurations, and recommended care in choosing leaders in peer-led antismoking programs, so that all types of students would have someone to identify with. However, recruitment of "Fonzie"-type students is a problem due to after-school commitments, reluctance of teachers to excuse them from class or allow exposure to younger impressionable students, and the lack of nonsmokers in this group. Also, the adolescent smoker is likely to engage in other early health-compromising behaviors such as alcohol and drug use and early sexual activity. Thus, smoking education should promote healthy behavior in addition to a smoke-free environment for the school society as a whole. See also 83-1283.

84-0560. Grabowski, J.; Bell, C. S. (Editors). **Measurement in the Analysis and Treatment of Smoking Behavior.** U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Pub. No. (ADM) 83-1285, 1983, 131 pp.

Issues, methods, and technologies in smoking research design and measurement are discussed, including an overview of smoking research strategies, and general guidelines that could increase validity, compatibility, and comparability of data across studies. Specific topics addressed are: (1) use of biologic fluids in assessing smoke exposure; (2) measurement issues in basic behavioral and physiological effects, and patterns of nicotine self-administration; (3) analysis of reinforcement by varying smoke component concentrations; (4) physical indicators of actual cigarette yields of tar and nicotine; (5) measurement of smoking topography in the natural environment; (6) smoking cessation data collection and questionnaire design; (7) standards for evaluation of smoking risk; and (8) survey and evaluation of smoking prevention activities among children and adolescents.

84-0561. Greiber, R.; Vargas, N.A. **Habito de Fumar en Estudiantes y Profesionales de la Salud. [Smoking Habits Amongst Students and Professionals of the Health Sciences.]** *Revista Medica de Chile* 110(5): 473-478, May 1982, Spanish.

A survey of 496 students and 336 professionals in health-related fields in Santiago revealed that 48.8 percent were smokers, with no sex differences. A higher prevalence was found among medical students (51.4 percent), particularly in their last year (72.5 percent). In the professional group, the highest rates were among midwives (56.0 percent) and medical technologists (52.0 percent); 38.1 percent smoked >10 cigarettes/day and 11.9 percent consumed >20 cigarettes/day. The smoking habit started early in life, 89.9 percent before 20 years of age and 39.4 percent under age 16. Friends and family influences were the most important factors related to smoking onset while pleasure was related to persistence. Consumption was higher in social gatherings and during working hours; 25 percent smoked all day. Cough, with or without sputum, was the most frequent complaint; 46.3 percent of smokers had no complaints. Over 90 percent of subjects were in favor of antismoking campaigns. (Auth. Abs. Mod.)

84–0562. Gust, S. W.; Pickens, R. W.; Pechacek, T. F. **Recording Puff Volume in Smoking.** *Behavior Research Methods and Instrumentation* 15(3): 341-343, June 1983.

A technique for recording puff volume, that uses a linear measure of flow rate and does not change draw resistance, is described. The subject places a cigarette mounted in a holder into one end of a plastic cylinder attached to a pneumotachograph. The pressure change in the pneumotachograph resulting from puffing is converted to an electrical signal by a differential pressure transducer and recorded on a dynograph. At the same time, an integrator coupler mathematically integrates the flow rate over time to give puff volume. Because air is drawn first through the pneumotachograph and then through the cigarette, the pneumotachograph remains unaffected by smoke. Calibrated samples showed a high degree of linearity ($r=0.99$) with this method. The drawbacks and advantages of other procedures for measuring puff volume are reviewed.

84–0563. Haley, N. J.; Axelrad, C. M.; Tilton, K. A. **Validation of Self-Reported Smoking Behavior: Biochemical Analyses of Cotinine and Thiocyanate.** *American Journal of Public Health* 73(10): 1204-1207, October 1983.

Biochemical determinations of plasma and salivary cotinine and thiocyanate in 12 smokers and 18 nonsmokers indicate that cotinine is better suited than thiocyanate (1) to determine smoking status in large scale epidemiologic studies, and (2) to follow alterations in smoking behavior over periods of time. Salivary cotinine is a reliable alternative to plasma for validation of smoking status and for following changes in daily smoking patterns. (Auth. Abs. Mod.)

84–0564. Hallett, R. **Intervention Against Smoking and Its Relationship to General Practitioners' Smoking Habits.** *Journal of the Royal College of General Practitioners* 33(254): 565-567, September 1983.

Responses from 342 general practitioners in London and Kent indicated that doctors who smoked cigarettes (13 percent of the sample) were less likely to advise or help their patients stop smoking than those who smoked a pipe or cigars or who did not smoke at all. A postal survey was carried out in June and July 1980 of 500 general practitioners (80.5 percent response from Kent, 60.3 percent response from London) to determine if there was any relationship between their smoking habits and reported intervention against smoking among their patients. Data also revealed a general practitioner who was an ex-smoker was as likely as a never smoker to intervene (a hopeful sign), and that the percentage of doctors who smoke continues to decline. It is hoped that the trend will continue and that routine intervention against smoking will become progressively more widespread.

84–0565. Hansen, W. B. **Behavioral Predictors of Abstinence: Early Indicators of a Dependence on Tobacco Among Adolescents.** *International Journal of the Addictions* 18(7): 913-920, 1983.

High school-aged smokers ($n=76$), from two California school districts, were surveyed regarding recent attempts to give up cigarette smoking and their smoking behavior (duration of habit, frequency of use, and regularity of use). For smokers who had attempted to quit (72 percent), a stepwise multiple regression analysis was performed. Questionnaire results showed that (1) students smoked an average of 15 to 20 cigarettes/day; (2) had smoked between 2 and 3 years; (3) 42 percent reported the same amount from day to day; (4) 46 percent reported some variation; (5) 12 percent reported large variations in day to day smoking; and (6) 65 percent had tried to quit only once. While 35 percent had attempted cessation more than once, 65 percent of the subjects relapsed within 1 month, and 82 percent within 6 months. The average number of cigarettes/day and the variability of smoking significantly predicted the length of time subjects were able to remain abstinent; those who relapsed early were characterized as relatively frequent, regular smokers. Duration of habit did not correlate with abstinence. Data provide evidence that dependence on tobacco is established relatively early in the lives of cigarette smokers. The finding that regularity, duration, and frequency of use were independent dimensions of smoking was unexpected, and suggests that there are several dimensions that may be useful in the categorization of young smokers and that may have implications for prevention programs. (Auth. Abs. Mod.)

84–0566. Hatch, J. P.; Bierner, S. M.; Fisher, J. G. **The Effects of Smoking and Cigarette Nicotine Content on Smokers' Preparation and Performance of a Psychosocially Stressful Task.** *Journal of Behavioral Medicine* 6(2): 207-216, June 1983.

While all subjects experienced smoking as relaxing and reported a stronger desire to smoke under negative (compared with positive) affective states, neither the smoking ritual nor the nicotine content of cigarettes smoked significantly affected any of four behavior dimensions studied before or during a psychosocially stressful task (extemporaneous speaking). Peripheral autonomic, electrocortical, cognitive, and overt motor responses were evaluated. Thirty smokers (20 or more cigarettes/day) were divided into three groups and, while preparing to speak, either did not smoke, smoked a low-nicotine cigarette, or smoked a high-nicotine cigarette. Results are discussed in terms of previous psychophysiological studies utilizing different types of stressors and behavioral indices of emotion. (Auth. Abs. Mod.)

84–0567. Hegazy, N. M.; Rizk, S. A.; Massoud, A.A.E. **Medical Students' Attitudes Towards Smoking.** *Journal of the Egyptian Medical Association* 65(9/12): 379-384, 1982.

Of 280 randomly selected male medical students at a Cairo university surveyed for smoking habits and related attitudes, 51.7 percent (n=160) had been smokers for 5 to 10 years and 70 percent consumed more than 20 cigarettes/day. While 95 percent were aware of health hazards, they continued to smoke. The most frequent reason for smoking initiation was imitation of parents or friends; also correlated with increased probability were death of at least one parent and parents' university education. Health authorities, educators, social workers, and psychologists are urged to develop methods to change community attitudes toward smoking.

84–0568. Henningfield, J. E. **Measurement Issues in Cigarette Smoking Research: Basic Behavioral and Physiological Effects and Patterns of Nicotine Self-Administration.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 27-38.

Data collected from studies that utilized two general nicotine self-administration strategies provide an experimental basis for assessing the role of the multiple factors involved in cigarette smoking. The two strategies were a cigarette smoking paradigm and an intravenous nicotine self-administration paradigm. Some of the factors discussed are: (1) problems of measurement in tobacco research, e.g., the nature and quantity of constituents in any given puff of smoke as a function of multiple factors, including cigarette constitution, puff topography, and inhalation characteristics; and (2) the equivalence of i.v. nicotine to tobacco smoke, a factor in determining the role of nicotine in the dependence process. Results suggest that measurement issues can be surmounted by systematically applying the methods of behavioral pharmacology and biological assay procedures. The use of multiple measures help resolve issues arising from any single measure. It is concluded that cigarette smoking is appropriately categorized as an instance of drug dependence in which nicotine is the key pharmacologic mediator of the behavior (but only one of the many components involved in the acquisition, maintenance, and elimination of the dependence process).

84–0569. Hyllienmark, G. **Rokning, Rokfrihet och Konsroller. [Smoking, Freedom From Smoking and Sex Roles.]** *Tobaken Och Vi* 28(3): 16-22, 1983, Swedish.

A hundred years ago in Sweden, smoking represented enjoyment for strong and economically successful men; less wealthy males enjoyed occasional cigars at celebrations. Smoking among women was considered immoral and was permitted only by women who were not dependent on men. In the 1920s, more women worked, and those with jobs began to smoke. By the 1940s, 75 to 80 percent of men smoked, regardless of professional status, and 52 percent of women smoked, with the majority of nonsmokers in low-status jobs. Among those born in the 1930s and 1940s, women's cigarette consumption had caught up with that of men: only 16 to 26 percent had never been daily smokers. Nonsmoking was most common in men and women with high-status jobs. In those born after 1950, smoking has declined for economic, social, and health-related reasons. Even so, cigarette use is still increasing in women with lower educational and socioeconomic status, partly because antismoking information is usually given by doctors, who are high-status males, and is viewed as condescending and an imposition of morality. Means of overcoming this resistance are suggested.

84–0570. King, A. C.; Scott, R. R.; Prue, D. M. **The Reactive Effects of Assessing Reported Rates and Alveolar Carbon Monoxide Levels on Smoking Behavior.** *Addictive Behaviors* 8(3): 323-327, 1983.

Alveolar carbon monoxide (COa) measurements confirmed the reliability of self-reported cigarette consumption and produced a reactive effect on smoking behavior. Participants in a Veterans Administration Smoking Treatment Program (52 men, 4 women), who had smoked an average of 31.5 years, attended two treatment sessions within 2 weeks at which consumption was reported and COa measurements taken; subjects were not asked to reduce consumption. Significantly lower smoking rates were reported at the second session (from a mean of 30.24 to 24.25 cigarettes/day) and a concomitant drop in COa was found. Smoking rates and COa levels were significantly correlated. Tar and nicotine content and carbon monoxide output correlated with each other but not with COa. The estimated time since the last cigarette did not correlate with any other variable. It is suggested that use of a simple assessment procedure can produce a reactive effect on smoking behavior.

84–0571. Kozlowski, L. T. **Physical Indicators of Actual Tar and Nicotine Yields of Cigarettes.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis on Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 50-61.

Problems inherent in the currently used standards of average smoking patterns and the deficiencies of smoking machine assays are explored. These problems center around interindividual variability and interpersonal inconsistency in puff number, puff duration, puff volume, and vent-blocking behavior. As an alternative to the standard assay for determining smoke exposure, analysis of the tracks of tar stains left in cigarette filters is suggested. Photographic examination of stain patterns reveals hole-blocking, and color-matching techniques to grade stain intensity would indicate tar yield. The origins of the standard regimen, in the laboratory of the American Tobacco Company in the 1930s, are traced.

84–0572. Lando, H. A. **Data Collection and Questionnaire Design: Smoking Cessation in Adults.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 74-89.

The construction of a simple and convenient questionnaire, with adequate reliability and validity, which could be used to standardize data collection by researchers and clinical practitioners, is discussed. Previous standards have provided little incentive for implementation, especially in public service settings. An excellent starting point for formulating recommendations for data collection was provided by the guidelines from the National Center for Health Education (NCHE). These stringent guidelines have been adopted by many public service and proprietary agencies. The American Lung Association (ALA) provides leaders with standard forms and questionnaires, and followup guidelines. The NCHE guidelines are realistic and do not appear to impose an excessive response burden, although they fail to provide for independent validation of self-reported smoking. The issues related to smoking measurement are reviewed, noting that correlates of smoking behavior are a promising possibility. The ALA questionnaires are useful starting points for smoking cessation studies, but their predictive powers might be improved if subjects were allowed a greater range of choices. Other measures of abstinence, withdrawal, and physiological correlates are also discussed. Additional issues in data collection and clinic evaluation, including long-term followup and recidivism, are also reviewed. It appears possible that a relatively sophisticated assessment of smoking patterns can be achieved at fairly minimal cost.

84–0573. Lichtenstein, E.; Grabowski, J.; Bell, C. S. **Issues in the Measurement of Smoking: Summary and Discussion.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 105-112.

Progress in measurement of smoking has been very substantial and is expected to continue. In the meantime, material in this volume provides an overview of strategies and methods for measurement of smoking behavior. Despite its seeming simplicity, smoking is a complex biobehavioral process and cannot be delineated by a single question or indicator. Both the laboratory investigator and the field researcher should profit from the material, which is organized under three major areas of concern: (1) determination of current smoking or abstinence; (2) dosage or exposure to tobacco (nicotine), its metabolites, and tobacco combustion products; and (3) characteristics of tobacco users that entail examination of physiological and pharmacological correlates of behavioral patterns and delineation of relationships. Topics are discussed in a general way, with some attention paid to cost—benefit considerations.

84–0574. McNair, E.; Bryson, R. **Effects of Nicotine on Weight Change and Food Consumption in Rats.** *Pharmacology Biochemistry and Behavior* 18(3): 341-344, March 1983.

Nicotine withdrawal produced significant ($p<0.05$) weight gains, and nicotine administration resulted in inhibition of weight gain in rats. Three-month-old Sprague-Dawley rats, 12 males and 12 females, were injected with either 0.2, 0.4, or 0.6 mg nicotine/kg body weight, three times/day. Half the rats received nicotine subcutaneously for 3 weeks followed by saline for 3 weeks; treatment was reversed for the other half. A significant ($p<0.05$) sex X drug X time interaction demonstrated that food consumption increased when nicotine was discontinued and decreased when nicotine was administered. Analyses showed the effects were strongest at the 0.6 dosage, and larger effects were obtained among males than in females. (Auth. Abs. Mod.)

84–0575. Millar, W. J.; Van Rensburg, S. **Tobacco Use Among Students in the Northwest Territories, 1982.** Health and Welfare Canada, Health Services and Promotion Branch, and Government of the Northwest Territories, Ottawa, 1983, 45 pp.

Data from a 1982 survey showed that smoking rates for students in the Northwest Territories are among the highest recorded for a school population (49 percent of boys and 53 percent of girls). Smoking onset is earlier among Inuit and Indian/Metis youth than among nonnatives, and earlier in girls than in boys. Prevalence of regular smoking is also higher in girls. In those aged 15 to 19, 61 percent of Inuit, 49 percent of Indian/Metis, and 47 percent of nonnative girls were regular smokers compared with 55, 39, and 36 percent of boys, respectively. In addition, girls are more likely to smoke more cigarettes; 20 percent of girls smoked over 60 cigarettes/week compared with only 17 percent of boys. Total consumption is highest among nonnatives. While approximately 9 percent use chewing tobacco or snuff, use tends to be more prevalent among Inuit and Indian/Metis boys aged 10 to 14. After age 14, usage levels decline for Indian/Metis youth but remain stable for Inuit youth. The baseline measures established will be used to develop health education programs and assess their effectiveness.

84–0576. Miller, R. E.; Cappiello, L. A. **Relationships Between Inmates' Past Drug Practices and Current Drug Knowledge**

and Attitudes. *International Journal of the Addictions* 18(6): 881-890, 1983.

Of 200 male inmates who volunteered for a drug knowledge/practice study, 76 percent reported being daily smokers, 11.5 percent had never tried cigarettes, and the remaining 12.5 percent were ex-smokers, experimenters, or smoked no more than once a week. A questionnaire was used to measure past drug practices and current drug knowledge and attitudes. Data analysis examined relationships between the variables. Nicotine ranked third (88.5 percent ever-users), with alcohol as the most used (94 percent), and marihuana listed as second (92 percent). PCP ranked last (45 percent). Significant positive correlations were recorded between drug knowledge and drug practices, and between drug knowledge and drug attitude scores. However, a significant negative relationship was noted between drug practices and drug attitude scores. Additional findings about inmates' demographic characteristics and drug variable measures are included. Implications are discussed for the development of drug education programs in jails and prisons. (Auth. Abs. Mod.)

84–0577. Moreton, W. J.; East, R. **Identifying Attitude Factors Associated With Smoking Cessation.** Kingston Upon Thames, England, Kingston Polytechnic, 1983, 97 pp.

Attitudinal and belief changes that correlated with successful smoking cessation and relapse were explored in 228 members of five stop-smoking groups. Abstinence among women was most closely associated with expected approval from other people. For men, personal gains from stopping were better indicators of success, although perceived social pressure prevented later relapse. Both men and women were more successful if beliefs about being miserable, craving cigarettes, and the effects of stress were changed. Also, women who changed expectations about being irritable and bored were more often successful. Contrary to expectations, the abstinent often lowered expectations about health gains, with two exceptions: (1) men with increased perceptions of heart attack risk reduction were more often successful; and (2) women with increased health-related fears were less likely to relapse. Implications of the findings for mass media antismoking campaigns and as reinforcers of abstinence after cessation are discussed.

84–0578. Nakamura, R. M.; Lescault, C. M. **Health Behavior Survey of California School Health Educators.** *Journal of School Health* 53(9): 557-560, November 1983.

A health behavior survey of California School Health Association (CSHA) members (6 males and 34 females, aged 30 to 60 years) showed that 15 percent smoked cigarettes, 2.5 percent smoked cigars, and there were no pipe smokers. Overall, a low percentage of individuals exhibited drug- and risk-taking behaviors, and a high percentage practiced proper nutritional habits and daily exercise. The high percentage of individuals maintaining good medical profiles and overall sound health practices suggests that CSHA members are potentially effective role models in the school health curriculum.

84–0579. Needle, R.; McCubbin, H.; Lorence, J.; Hochhauser, M. **Reliability and Validity of Adolescent Self-Reported Drug Use in a Family-Based Study: A Methodological Report.** *International Journal of the Addictions* 18(7): 901-912, 1983.

Survey results from 155 Minneapolis junior and senior high school students indicate that self-reports of alcohol, tobacco, or marihuana use are generally reliable. A pretest identified 73 users and 82 nonusers, who were randomly assigned to one of four groups: (1) group I, who completed a questionnaire at school and 2 weeks later at home with their parents; (2) group II, who completed the questionnaire at home with their parents and again 2 weeks later at school; and (3) two control groups that took the test either at school (in small groups) or at home (without parental participation). Consistency values for group I between school and home completion of the test were 95.9, 97.3, and 97.3 percent for self-reported lifetime, 12-month, and 30-day cigarette consumption, respectively. Although no direct objective validation of the self-report measures was used, evidence suggests that the setting in which respondents complete questionnaires does not result in systematic reporting bias.

84–0580. Nichol, N.; Russell, J. **A Study of the Smoking Habits of Pregnant Women and Their Attitudes to Smoking in Hospital.** *Health Bulletin* 41(5): 233-237, September 1983.

Responses (n = 181) to questionnaires distributed to consecutive female patients attending prenatal and booking clinics at an Edinburgh hospital indicated that 38 percent (n = 68) were smokers and 62 percent (n = 113) were nonsmokers. Nearly 60 percent of the respondents under age 25 were smokers, greatly exceeding the national average. Data show that 42 percent of all Scottish women were smokers in 1980. Half the responses indicated that patients should not be permitted to smoke in hospitals, compared with 34 percent indicating that smoking should be allowed. Of smokers, 6[illegible] percent felt they should be permitted to smoke, compared with only 15 percent of nonsmokers. Of all respondents, 72 percent (62 percent of smokers and 78 percent of nonsmokers) preferred separate wards for smokers and nonsmokers; 70 percent (20 percent of smokers) preferred a nonsmoking ward; and 75 percent indicated that hospital visitors should not be permitted to smoke.

84–0581. Nil, R.; Woodson, P.; Baettig, K. **Der Raucher, Sein Rauchverhalten und Seine Rauchmotivation. [The Smoker, His Smoking Behavior and His Motivation for Smoking.]** *Psychologie* 41(2): 217-229, 1982, German.

Nicotine is probably the only pharmacologic reason for smoking, and appears to both stimulate and tranquilize, depending on the situation. Nonpharmacologic motivators for smoking (heredity and environment) appear to have additive, but separate, effects. For instance, there are more smokers among those in the lower social and educational classes, under age 50, in industrialized and urban areas, and among the unmarried, divorced, and separated. Smokers are more likely to be extroverts, and tend toward alcohol and drug consumption, and other "exotic" behavior. The connection between neuroticism and cigarette use is controversial. However, highly neurotic smokers take in greater amounts of nicotine and are less able to abstain from smoking. It is noted that, regardless of type and number of cigarettes smoked, nicotine intake appears to be equally regulated.

84–0582. O'Rourke, A. H.; O'Byrne, D. J.; Wilson-Davis, K. **Smoking Among Schoolchildren.** *Journal of the Royal College of General Practitioners* 33(254): 569-572, September 1983.

A questionnaire survey in 1981 of over 5,000 postprimary school students in Dublin showed that smoking rates in boys have changed little since 1971, but have increased among girls. Overall, 34 percent of boys and 26 percent of girls were regular smokers (consumed at least one cigarette/week). In 1970, 35 percent of boys but only 18 percent of girls smoked. The increase was greatest at ages 13 and 14, and by age 17 more girls than boys had experimented with cigarettes. Only 26 percent of all males and 37 percent of all females in the sample had never tried cigarettes. Peer smoking was significantly associated with the subject's smoking habits. Sibling smoking was also positively correlated, but parental smoking showed little influence. Over half the smokers said they would like to quit. Social and sporting reasons for cessation were cited far more frequently than those concerning long-term health effects.

84–0583. Pickens, R. W.; Gust, S. W.; Catchings, P. M.; Svikis, D. S. **Measurement of Some Topographical Aspects of Smoking in the Natural Environment.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 62-73.

A small, portable, solid-state device that can be used in the natural environment to measure individual puff durations, interpuff intervals, cigarette durations, and intercigarette intervals, is described. Computer software has been developed to facilitate its use, and results of studies on its accuracy and reliability are reported. Previous studies of smoking topography have been largely confined to the experimental laboratory, but with the ambulatory smoking recorder, a high level of agreement was found between subjects' self-report records and recorder data. Results suggest that situation as well as mood may influence smoking patterns in the natural environment. When smoking topography was compared for the most frequently occurring situations and mood states, large differences were found, suggesting that factors affecting smoking may not be amenable to laboratory control. In addition, results from natural environment studies could be used to validate results of laboratory studies, and determine whether such findings will generalize to the natural environment. Such information might also help explain differences in success rates of various groups of smokers who try to quit. It might also provide data relevant to other drug dependencies, where studies in the natural environment are more difficult to conduct.

84–0584. Roe, F.J.C. **Any Questions? (Letter).** *British Medical Journal* 287(6401): 1291, October 29, 1983.

While pipe smoking has been found to be less associated with lung cancer, chronic obstructive respiratory disease, and coronary heart disease than cigarette smoking, the reasons are not clear. The method of smoking (pipe vs. cigarette) and the way a smoker manipulates smoke (inhalation) to experience nicotine-dependent effects may be as important as the way the tobacco is cured (cigarette tobacco is generally flue-cured; pipe tobacco sun-dried). Other differences between pipe smokers and cigarette smokers, including temperament and lifestyle, are also discussed.

84–0585. Rose, J. E. **Analysis of Reinforcement by Varying Smoke Component Concentrations.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 39-49.

A new methodology for studying the reinforcing actions of nicotine explores variations in nicotine preference while the concentration of nicotine in each puff of smoke is under the subject's control. Using a smoke-mixing apparatus, nicotine concentration can be adjusted and optimal delivery selected. To measure variations in preference resulting from smoking abstinence and satiation, 20 smokers (12 males, 8 females, average age 38 years) were tested, and results under both conditions were compared. Subjects preferred significantly higher nicotine concentrations in the deprived condition, confirming nicotine's role as a desired ingredient in cigarette smoke. Although primarily designed to study nicotine preference, the smoke-mixing methodology can be applied to the investigation of other factors important in smoking reinforcement. For example, by placing cigarettes of equal nicotine delivery (but low- and high-tar delivery) in the smoke mixer, preference for tar concentration could be measured. In a similar manner, preference for any constituent of tobacco smoke might be explored.

84–0586. Rosen, T. J.; Shipley, R. H. **A Stage Analysis of Self-Initiated Smoking Reductions.** *Addictive Behaviors* 8(3): 263-272, 1983.

Multiple regression analyses associated three distinct stages of self-initiated smoking reduction with three separate predictor scales measured by multi-item tests, indicating the value of both stage analysis and smoking cessation treatment focused according to stage. The three stages of cessation are decision, initial control, and successful maintenance, and the three predictor scales are desire to stop, self-esteem, and internal locus of control combined with a strong desire to stop. Analyses were based on three different samples of Veterans Administration health fair volunteers: 61 subjects in a pretest sample, 51 in the followup sample, and 23 in the sample of those who had tried to quit. Of the 51 subjects followed up at 2 months, four had quit but had not remained abstinent, while overall smoking rate was 85.5 percent of baseline (23.4 cigarettes/day). For the 23 who tried to reduce, the mean lowest percent of baseline (31.7, SD=24.1) and the mean relapse (76.8, SD=52.1) did not correlate, permitting attempts to predict both. It is indicated that (1) stage analysis led to the observation of interstage differences that would have been hidden by a global analysis; and (2) treatment should address the immediate needs and concerns of prospective quitters, particularly those who are unsuccessful when left to their own devices.

84–0587. Sachs, D.P.L. **Smoking Habits of Pulmonary Physicians. (Letter).** *New England Journal of Medicine* 309(13): 799, September 29, 1983.

A sampling of the 2,207 pulmonary physicians practicing in the United States (n=594 or 27 percent) attending the 1983 American Thoracic Society annual meeting showed that 4.6 percent were cigarette smokers, compared with 17 percent of internists who reported smoking in 1975. Since the 1975 survey did not provide information regarding medical specialty, it is unknown whether this decline represents a difference in prevalence of smoking between pulmonary specialists and internists, or a downward trend among pulmonary physicians. However, either interpretation (considered with data showing smoking prevalence among internists of 60 percent in 1945 and 22 percent in 1967) indicates compliance with the expectations of 80 percent of American patients who believe that their physicians should be nonsmoking exemplars.

84–0588. Saucier, J.-F.; Ambert, A.-M. **Parental Marital Status and Adolescents' Health-Risk Behavior.** *Adolescence* 18(70): 403-411, Summer 1983.

Both boys and girls from broken families smoked significantly more than those from intact families, and adolescents from legally broken families smoked more than those who had suffered the death of a parent. Surveys in 1977, of 4,539 students in high school and the first 2 years of college, found that the Montreal Island students from separated/divorced homes engaged in more health-risk behaviors, especially smoking and not using seat belts. Differences in intemperate drinking behavior were not significant between the groups, although students from intact families tended toward lower scores. Results for adolescents from widowed families were mixed. It is noted that both separated mothers and fathers smoked more than those in intact marriages, and smoking among their children may thus be a function of role modeling. Also, nonintact families are less ordered and health-risk behaviors such as smoking are more easily overlooked.

84–0589. Schenk, J. **Die Personlichkeit des Drogenkonsumenten. [The Personality of the Drug Consumer.]** Gottingen, Germany, Verlag fur Psychologie, Dr. C. J. Hogrefe, 1979, 200 pp., German.

Smoking habits are considered in an investigation of the research issues surrounding personality and drug use. The first four chapters review the problems inherent in drug use research, describe the battery of tests utilized, and present the results of the various tests. The final four chapters report the findings of a sample study of drug consumption (nicotine, alcohol, cannabis, hallucinogens, opiates, and others) among army recruits. The patterns of consumption of these drugs are analyzed, and attempts are made to correlate these patterns with personality factors. The controversial connections between smoking and extroversion, and smoking and neuroticism are explored. It is noted that tobacco is the most frequently consumed substance among the recruits.

84–0590. Schinke, S. P.; Gilchrist, L. D. **Survey and Evaluation Methods: Smoking Prevention Among Children and Adolescents.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 96-104.

Recommendations on common parameters of survey and evaluation methods of researching tobacco use among American children and adolescents are discussed, noting that more precise benchmarks of these age groups will facilitate mutually advantageous investigations. The peculiarities of nonadults who smoke are reviewed, and methods of determining tobacco consumption in the target age groups are compared and contrasted in three main areas: (1) defining childhood and adolescence; (2) disparate smoking patterns; and (3) weaknesses of self-report. Seven categories of youthful tobacco smoking are defined that are responsive to any youthful age group and tobacco substance, and that permit movement from one group to another as smoking behavior changes. When scientists adopt the seven groupings, normative data can be amassed and exchanged.

84–0591. Schoonmaker, R. B., Jr. **A Teenage View of Cigarette Smoking.** *Connecticut Medicine* 47(11): 705-707, November 1983.

Of 1,614 sophomore Middlesex County high school students who completed a questionnaire, 18 percent of the males and 22 percent of the females smoked at least one cigarette/week. The attitudes of the students toward cigarette use were assessed and analyzed according to sex and smoking status of the respondents. Cigarette use was significantly related to the smoking habits of parents, older siblings, and friends. Of the nonsmokers, 94 percent stated it was unlikely they would be smoking at time of graduation, while 74 percent of the smokers said they had no intention of quitting. Half the students felt that most adults smoked, while 40 percent of the nonsmokers vs. 58 percent of the smokers felt that most high school students smoked. A large group of the sophomore smokers did not think smoking improved their self-image.

84–0592. Sheehan, T. P. **Children and Smoking. A Review.** *Australian Family Physician* 12(9): 685-687, September 1983.

Forty percent of smokers in the United States start before the age of 9. Average ages are 10 and 11 years for boys and girls, respectively. A 1979 South Australian primary school survey showed that 10.3 percent of boys smoked regularly before age 12; 20 percent of boys and 31 percent of girls in Western countries begin smoking by age 13, and 28 and 32 percent, respectively, by age 14. While children who smoke are more likely to have parents or older siblings who also smoke, peer group pressure is the most important influence on the older child. Male adolescents associate smoking with masculinity; females relate the habit to sophistication. Children most likely to become smokers have poor academic records, are underachievers, impatient to become adults, and more rebellious. Permissive parents, urban residence, lower socioeconomic status, and single parent households are also associated with children "at risk." Antismoking messages should be positive, stressing the benefits of not smoking (i.e., attractiveness, fitness, good health), and antismoking strategies should promote awareness of immediate and medium-term physiological and psychological smoking effects (i.e., blood pressure, content of inhaled air, attractiveness, low birth weight and respiratory problems in neonates, deleterious effects in sperm). Positive peer pressure can be developed through attractive nonsmoking peer and exemplar models, and children can be educated to resist advertisers' gimmicks and encouraged to participate in activities that expose smoking disadvantages (i.e., sports). A list of Australian antismoking centers is provided.

84–0593. Spealman, R. D. **Maintenance of Behavior by Postponement of Scheduled Injections of Nicotine in Squirrel Monkeys.** *Journal of Pharmacology and Experimental Therapeutics* 227(1): 154-159, October 1983.

Responding by squirrel monkeys was maintained by a schedule of i.v. nicotine postponement. Each lever-pressing response postponed an impending injection for 60 sec; in the absence of responding, injections occurred every 20 sec. The lowest dose of nicotine (10 μg/kg/injection) maintained rates of responding that were only slightly higher than those maintained by saline. Intermediate doses (30 or 56 μg/kg/injection), however, maintained rates and patterns of responding that were similar to those maintained by electric shock under an identical schedule of postponement. At the highest dose (100 μg/kg/injection), nicotine disrupted responding and induced vomiting in each monkey. Presession treatment with mecamylamine (1.0 mg/kg i.m.), a nicotinic antagonist with both central and peripheral effects, shifted the nicotine dose—response curve to the right by about 1 log unit and blocked nicotine-induced emesis. In contrast, presession treatment with hexamethonium (1.0 or 10.0 mg/kg i.m.), a nicotinic antagonist with primarily peripheral effects, did not produce an overall rightward shift in the nicotine dose—response curve, although it did reduce the rate of responding maintained by the intermediate doses of nicotine and blocked emesis. Neither mecamylamine nor hexamethonium systematically altered responding maintained by electric shock under the schedule of postponement. The results show that schedule-characteristic performances can be maintained by postponement of i.v. injections of nicotine and that the maintenance of responding under the postponement schedule depends largely on the actions of nicotine in the central nervous system. (Auth. Abs.)

84–0594. Stepney, R. **Why Do People Smoke?** *New Society* 65(1080): 126-128, July 28, 1983.

When viewed as drug use, smoking is difficult to understand since there is no euphoria, no distortion of perception, no oblivion, nor any immediate ill effects, such as a hangover. There is some evidence, however, that nicotine can activate the "reward centers" in the brain, producing both stimulant and depressant effects, shown to be dose-related and dependent on the smoker's personality and circumstances. Some smokers may start out using nicotine, but end up as addicts who smoke to maintain nicotine levels. Since no society has been known to voluntarily give up widely used psychotropic substances, it may make sense to allow people to get nicotine in ways that do not involve smoking, such as (1) an inhaler similar to that used by asthmatics; (2) a "noncombustible" cigarette, which would deliver a metered dose of nicotine vapor; or (3) nicotine chewing gum. The personality of a smoker may differ from that of a nonsmoker due to heredity or the environment, and other factors may also be involved, but despite health warnings, they stick to cigarettes that produce the dose of nicotine sufficient to have the desired effect on the brain. Most smokers inhale, because it is the most efficient way of getting what is desired.

84–0595. Tsakraklides, V.; Mentis, A.; Efstathiou, P.; Trichopoulos, D. **Tobacco Smoking by Young Adult Males in Greece.** *International Journal of Health Education* 2(2): 26-30, July 1983.

In a survey of 6,650 Greek soldiers (aged 20 to 29), 4,729 (71 percent) reported regular smoking, 1,761 (27 percent) were nonsmokers, and 160 (2 percent) were ex-smokers. Data also show that (1) prevalence of smoking was inversely related to level of education; (2) the percentage of physicians who smoke was lower than for other scientists (60 percent compared with 68 percent, respectively); (3) physicians began smoking at a later age than those with less education; (4) the percentage of smokers from urban and rural areas was the same (72 percent), compared with only 68 percent for those from semi-urban areas; (5) smoking initiation was related to the presence of smokers in the family; (6) the large majority of the soldiers believe that smoking is hazardous to nonsmokers as well as smokers; and (7) more nonsmokers participated in sports than smokers. The smoking rate for young adult males in Greece is thus among the highest in the world, and may even be an underestimate of the rates for the total male population. It is concluded that unless urgent measures are taken to control the smoking epidemic, in the near future Greece may have one of the highest mortality rates for lung cancer.

84–0596. Van Reek, J. **Rookgedrag in Nederland van 1958-1982. [Smoking Behaviour in the Netherlands From 1958 to 1982.]** *Tijdschrift Voor Alcohol, Drugs en Andere Psychotrope Stoffen* 9(3): 99-103, 1983, Dutch.

Data from 23 Dutch smoking behavior surveys showed that male smokers decreased from 90 percent in 1958 to 41 percent in 1982. The percentage of both male and female ex-smokers markedly increased, as did cigarette consumption/female smoker. A stepwise logistic regression comparing smoking with background variables showed the following order of importance: sex, age, year, religion, social class, and degree of urbanization. As women's emancipation has been largely realized, and the influence of the Puritan ethic is small, it is expected that antismoking information will play a large and growing role in smoking reduction. (Auth. Abs. Mod.)

84–0597. Weeks, D. J.; Todd, S. P. **Cigarette Tar Cheat. (Letter).** *New Scientist* 99(1370): 431, August 11, 1983.

Further observations on the physical and social aspects of smoking that determine tar levels compared with levels established by smoking machines are presented. Among smokers in a local pub and a hospital cafeteria, the typical puff was of 2.5 to 3 seconds duration, occurring once every 45 seconds for females and once every 50 seconds for males; machines usually are set for a 35 ml puff for 2 seconds, once a minute. Almost 95 percent of smokers of vented filter cigarettes blocked the holes with their fingers. High consumption levels (5.4 cigarettes/hour) and smoking in groups suggest that much smoking is focused in particular, clearly defined situations that cannot be simulated by robot smoking. It is estimated that smokers receive more than twice the amount of tar as indicated in government tables.

84–0598. Wesnes, K.; Warburton, D. M. **Smoking, Nicotine and Human Performance.** *Pharmacology and Therapeutics* 21(2): 189-208, 1983.

Cigarette smoking is shown as helping to sustain performance on monotonous tasks and to produce absolute improvements in both the speed and the accuracy of information processing. Nicotine tablets have comparable effects, indicating that the drug is responsible for the improvements produced by smoking. These improvements can be explained by the increase in electrocortical arousal, produced by the action of nicotine on the ascending reticular cholinergic pathways. These effects of smoking on cognitive function, when considered together with the rapid absorption and elimination of nicotine, suggest a major role in the motivation for smoking. (Auth. Abs.)

84–0599. Wesnes, K.; Warburton, D. M. **Effects of Smoking on Rapid Information Processing Performance.** *Neuropsychobiology* 9(4): 223-229, October 1983.

Results indicate that smoking produces absolute improvements in rapid information processing performance. Two experiments are described that were designed to investigate the effects of smoking on the performance of a rapid information processing task. The task involved the detection of sequences of three consecutive digits of the same parity from a series of digits presented visually at the rate of 100/min. In the first experiment, smoking improved both the speed and accuracy of performance above rested baseline levels, the greatest improvement occurring with the highest nicotine and tar delivery cigarette. In the second experiment, smoking again improved the speed and accuracy of performance above baseline levels, while performance deteriorated over time after not smoking as well as after smoking a nicotine-free cigarette. Results are explained in terms of the action of nicotine on central cholinergic pathways. (Auth. Abs. Mod.)

84–0600. Wyshak, G.; Lawrence, R. S. **Health-Promoting Behavior Among Lawyers and Judges.** *Journal of Community Health* 8(3): 174-181, Spring 1983.

Survey results indicate that only 10 to 20 percent of judges and lawyers were smokers in 1979, compared with 38 percent of males in the general population. Data on health-related behaviors were obtained by questionnaire from lawyers and judges attending an annual meeting. Eighty percent of the lawyers and 90 percent of the judges reported being nonsmokers; nearly 60 percent of the judges aged 55 and over and 40 percent of those aged 40 to 54 were never smokers. A third of each age group were ex-smokers. Exercise and seat belt use were also more common in the two professions, although drinking habits and drinking problems, diet and weight consciousness, and prevalence of occupational stress and tension were similar to rates reported in the general population.

84–0601. Zoller, U.; Maymon, T. **Smoking Behavior of High School Students in Israel.** *Journal of School Health* 53(10): 613-617, December 1983.

Analysis of questionnaire responses showed that smoking behavior and attitudes among high school students (aged 14 to 18) in Israel are similar to those of students in the United States. To evaluate the recently implemented Israeli educational program "Smoking and Cigarette Smoke," 1,420 students in 22 schools were administered a 65-item questionnaire. At the 10th grade level, one-third of the boys were smokers; during 11th grade, the girls caught up and surpassed the boys' level. Nearly half (46 percent) of the students had never tried cigarettes, and only 3.8 percent reported consuming five or more cigarettes/day. The primary reason given for not smoking was that it was not pleasurable; health concerns were in second place, followed by parental influence. The majority of smokers tried cigarettes out of curiosity; other reasons included "felt like trying," showing off, peer influence, parental influence, pleasure, and rebelliousness.

See also, **84–0367, 84–0393, 84–0603**

SMOKING PREVENTION AND INTERVENTION

84–0602. Barwood, M.; Schmidt, F.; Hauknes, A.; Bjartveit, K.; Manderlier, T.; Viljoen, C.E.M.; Morris, K.; Rosales, V. J. **Medical Associations Step Up Activities to Counter Smoking. (Letters).** *New York State Journal of Medicine* 83(13): 1339-1344, December 1983.

Letters from seven national medical associations review the associations' current antismoking activities, obstacles to curbing smoking in the population, current cigarette consumption trends, and the impact of specific events, such as the National Day Without Tobacco (Belgium). The British Medical Association, the Forschurgsstelle fur Praventive Onkologie of West Germany, the Norwegian National Council on Smoking and Health, the Federation Belge des Chambres in Belgium, the Medical Association of South Africa, the New Zealand Medical Association, and the Philippine Medical Association responded to the request for information sent to associations in 45 countries. The British are pressing for stricter controls on the sponsorship of sporting and cultural events; the West Germans deplore the lack of government interest and unparalleled advertising expenditures by the tobacco industry; the Norwegians report on positive changes in consumption as a result of antismoking strategies; and the South Africans are calling for specific legislation to control advertising and limit sales. Commentaries from France and Hong Kong were not prepared in time for publication.

84–0603. Bell, C. S.; Grabowski, J. **Overview of Smoking Research Issues.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 1-5.

Data strongly supporting long-hypothesized relationships between tobacco and ill health have merged with behavioral/pharmacological reports clarifying similarities between tobacco and drug consumption, indicating future research needs, directions, and issues. Discrepancies or inconsistencies in research design and analysis must be identified. Issues include deficiencies in (1) controls and systematic design; (2) program validations developed in the research setting; (3) longitudinal data; and (4) attention to accepted theoretical guidelines or unifying conceptual premises. Further problems have included (1) small samples resulting in excessive data variability, and (2) insufficiently replicated measures when small samples have been appropriate. It is essential to develop researchers whose skills bridge the gap between traditional disciplines, permitting thorough analysis of biobehavioral disorders (especially those with multifaceted physiological and behavioral components). A search for similarities with other behaviors and forms of drug use might provide a useful model for analysis and for generating successful smoking prevention and cessation programs. Finally, there is a need to identify and delineate available guidelines for measurement in smoking research. These guidelines may be useful in prevention and treatment strategies, while systematic data collection would be an aid to maintaining consistency in data obtained.

84–0604. Bobo, J. K.; Gilchrist, L. D. **Urging the Alcoholic Client to Quit Smoking Cigarettes.** *Addictive Behaviors* 8(3): 297-305, 1983.

Survey data, from 311 alcohol treatment specialists staffing 23 inpatient facilities in Washington State, indicated that staff who described themselves as alcoholics and smokers were significantly less likely than nonalcoholic nonsmokers to urge smoking cessation efforts for alcoholics. Because cigarette smoking is highly correlated with alcohol abuse, and information on smoking cessation for alcoholics is scarce, personnel were asked to review a list of eight different time points, including "never," when an alcohol-involved adult could be urged to quit smoking. Respondents first selected those time points at which they favored and then those at which they personally encouraged smoking cessation. One year after the onset of sobriety emerged as the preferred time point. Responses were analyzed according to the treatment staff's self-reported alcohol and smoking status (39.5 percent considered themselves alcoholics, 35.3 percent rated themselves regular smokers). Nonalcoholics were more likely to want a cessation plan than alcoholics, but the difference between smokers and nonsmokers was not significant. Since studies have shown that consumption of either alcohol or tobacco may lead to consumption of the other, smoking cessation strategies should be designed for alcoholic clients. (Auth. Abs. Mod.)

84–0605. Dawley, H. H., Jr.; Baldwin, J. **The Control of Smoking: Smoking Rate in Designated Smoking and No-Smoking Areas.** *International Journal of the Addictions* 18(7): 1033-1038, 1983.

A comprehensive sign program (with peer pressure and staff requests) was found to effectively control smoking in designated hospital areas. Four times a day for 5 days, in three smoking and three nonsmoking areas, observations were made on smoking behavior of individuals during 10-minute intervals. Compliance with no smoking signs was 94.64, 94.40, and 98.92 percent for the ambulatory care area, the outpatient waiting area, and the no smoking lobby, respectively. In the smoking areas, the number of smokers ranged from 20.56 to 37.0 percent (in the smokers' lobby area). Thus, hospitals now have ways to protect patients from tobacco smoke pollutants.

84–0606. Dunn, A. G.; Dunn, P. **Letter From Perth. Puffability People: A New Way to Promote Health.** *British Medical Journal* 287(6397): 960-962, October 1, 1983.

The term "puffability" was coined for the percentage of predicted forced expiratory volume in 1 sec (FEV_1) for persons participating in spirometric testing at one of Perth's large public hospitals, one of several projects in November 1982 undertaken to promote awareness of the smoking problem. There are now about 70 volunteer Puffability People who are trying to counteract the tremendous lead held by tobacco companies in promoting their products. Virtually all are members by invitation (in late teens through early 30s) and are mostly doctors, medical students, nurses, other hospital staff, and athletes. While spirometric test results are crude, careful explanation appears, at least subjectively, to have great impact even on smokers within the normal range. However, most Western Australians will never receive a test (although several hundred people are tested during Thursday evening and Saturday morning sessions) and it is the Puffability People themselves who are really important. Effective promotional projects such as tee shirts, especially those created for children (Womble-size creatures) and antismoking poems and songs, are being planned. The main difficulty is finding funding, and it remains to be seen whether the new government, which has pledged itself to strong antismoking measures, is prepared to help.

84–0607. Egger, G.; Fitzgerald, W.; Frape, G.; Monaem, A.; Rubinstein, P.; Tyler, C.; McKay, B. **Results of Large Scale Media Antismoking Campaign in Australia: North Coast "Quit for Life" Programme.** *British Medical Journal* 287(6399): 1125-1128, October 15, 1983.

Findings of a three-community study support the use of the electronic media in health promotional appeals against smoking. Followup surveys were conducted in northern New South Wales, Australia, to determine the effectiveness of media intervention programs and media intervention plus community programs in altering lifestyle behaviors, including smoking, dietary fat intake, and exercise. Results of one aspect of the North Coast Healthy Lifestyle Program entitled "Quit for Life," which aimed at reducing cigarette smoking over a 2-year period, were analyzed. Declines in the prevalence of smoking in both test towns, ranging from 16 percent in younger men to 6 percent in older women, were significantly greater than the declines in the control town. The declines were more sustained over the 2-year period in the town subjected to the media campaign plus community programs. (Auth. Abs. Mod.)

84–0608. Hedrick, B. M.; Rubin-Lieberman, L.; Bruggeman, J. M. **UT Goes Cold Turkey: An Anti-Smoking Campaign at the University of Toledo.** *Health Education* pp. 37-39, September-October 1983.

Peer pressure, a significant factor in smoking initiation among 1 million teenagers annually, enables campus or schoolwide antismoking campaigns to more effectively reach teenagers than nationwide or community campaigns. A University of Toledo antismoking campaign, "UT Goes Cold Turkey," encouraged students to quit "Just for the Health of It." Factors identified as important for success were (1) adequate promotion and motivation by use of the school newspaper, local television and radio, movies (i.e., the Dick Van Dyke 1960's film *Cold Turkey*), and competitions between campus organizations; (2) enthusiastic, dedicated workers; and (3) adequate, accessible space (cafeteria or Student Union building). Positive peer pressure and successful techniques employed included pulmonary function testing by respiratory therapy students; film presentations of "The Feminine Mistake"; a reinforcement/encouragement campaign; a kissing booth ("kiss a nonsmoker and taste the difference"); cessation of cigarette sales at the Student Union building on Cold Turkey Day; and the "Crisis Closet," a room providing frustration-releasing activities. The Crisis Closet included a graffiti wall covered with newsprint, a slide—tape presentation of the American Lung Association's "Freedom From Smoking" self-help program, and other audiovisual aids, pamphlets, volunteers, and an atmosphere of support, encouragement, and relaxation rather than attack or condemnation. Recommendations for organizing a similar event include (1) obtaining a school organization or class to sponsor the activity; (2) defining objectives; (3) setting a date that allows time for adequate planning; and (4) obtaining consent and space needed. Organizers are advised to enlist an organization such as the lung association as a cosponsor, meet frequently and brainstorm, enlist other school organizations to provide special services, and choose health education material.

84–0609. Holleb, A. I. **A Minute of Your Time.** *Ca-A Cancer Journal for Clinicians* 33(5): 307-308, September-October 1983.

Gallup poll responses indicate that 71 percent of pack-or-more/day smokers would quit if their doctors advised them to do so, reflecting both the opportunity and responsibility of physicians to prevent, rather than simply treat, disease. Physicians must be able to advise patients regarding methods, and offer encouragement, guidance, referral to smoking cessation clinics, literature, and a minute of their time to discuss the importance of quitting. While giving up cigarettes isn't easy, evidence suggests that many smokers are motivated. The Department of Agriculture recently announced a 3 percent decline in domestic tobacco consumption, following a 1 percent drop in 1982. In a large sample of the American Cancer Society's current Cancer Prevention Study (CPS II), 38.7 percent of males and 21.1 percent of females are ex-smokers, compared with 17.3 percent and 5.6 percent, respectively, in 1959. Also, a 1981 survey showed that over 1 million participants in the 1980 Great American Smokeout were still abstinent 11 months later. Smokers need the moral support and sense of urgency that all physicians have the power to convey.

84–0610. Mason, J. O.; Lindsay, G. B. **A Positive Approach to Smoking Prevention and Cessation.** *Western Journal of Medicine* 139(5): 721-722, November 1983.

The proportion of adult Americans who smoke dropped from 42 percent in 1965 to 33 percent in 1982, representing a major achievement for physicians and public health educators. Current research in the United Kingdom demonstrates physicians' significant effect: (1) those who explained risks, distributed materials, and warned of followup influenced 17 times more patients to quit than control group physicians; (2) 39 percent of physician-advised chest clinic patients reduced smoking compared with 17 percent of nonintervention controls; and (3) high risk cardiovascular smokers attending three 15-minute smoking cessation/health education sessions with physicians attained a 35.5 percent 3-year abstinence rate, compared with 14.5 percent of nonintervention controls. While some physicians may hesitate to discuss behavior they perceive patients understand and have freely chosen, they should not overestimate patients' knowledge of health risks or underestimate desire or ability to quit smoking. Data indicate that successful cessation rates are significantly underreported: rates calculated only for hardcore smokers in formal programs who have tried unsuccessfully to quit are not representative of the higher cessation rates achieved by the general public. Successful physician counseling techniques and health education approaches for fifth and sixth graders are described.

84–0611. Nursing Times. **Helping People to Stop Smoking.** *Nursing Times* pp. 63-65, June 8-14, 1983.

In response to research indicating that nurses knew little more than the general public about smoking, the Health Education Council produced a package of antismoking teaching materials designed both to educate nurses and for their own use in educating patients. The stop smoking packages include fact sheets on smoking-related illnesses, statistics, and other health issues, and advice on teaching methods for hospital, community, and tutorial settings. Also, the guidelines address the nurse as a role model and encourage nonsmoking. An introductory competition was held for the best essay on use and efficacy of the materials.

84–0612. Ouellet, B. L.; Melia, P. **Toward a Generation of Non-Smoking Canadians.** *International Journal of Health Education* 2(2): 34-38, July 1983.

In 1981, the Canadian Department of National Health and Welfare initiated a major smoking prevention program, "Toward a Generation of Nonsmokers," or "Generation," which pursues the goal of developing a generation of nonsmokers by (1) promoting nonsmoking as a preferred behavior among children; (2) creating and maintaining high levels of awareness concerning the health consequences of smoking; (3) developing skills to resist social and peer pressures to smoke; (4) encouraging nonsmoking role models for children; (5) promoting smoking cessation among role models; and (6) promoting a nonsmoking social environment. In the first 5 years, priority will be given to startup of programs and social supports directed at critical stages of smoking onset. Institutions that reach children at different ages will be systematically drawn into the program, including nontraditional health education organizations such as youth clubs, youth-oriented retailers, and sports associations. For each developmental stage, the Generation program addresses key issues and intervention needs. It is noted that, despite a steady decline in smoking rates in Canada (from 43 percent in 1966 to 33 percent in 1981), most adults and youth perceive smoking as a majority behavior.

84–0613. Public Health Reports. **Smoking Control.** *Public Health Reports* Supplement: 107-116, September-October 1983.

The Public Health Service (U.S. Department of Health and Human Services), presents a summary of the smoking problem and its own goals and objectives for improvement of health via smoking prevention and cessation. Of the 17 goals listed, 10 are identified as priorities for Federal effort. These include reducing the proportion of adults that smoke to below 25 percent, and the proportion of smokers among youth to below 6 percent by 1990; dramatically increasing public awareness of the health risks from smoking; continuing epidemiological research and hazard surveillance; and strengthening of package warnings and labeling requirements. The role of the Federal Government in attaining these goals and a summary of Federal efforts are outlined. For each of the specific implementation steps, the agencies responsible and year of initiation are charted.

84–0614. Reinertsen, J. **Promoting Health Is Good Business.** *Occupational Health and Safety* pp. 18-22, June 1983.

An employee who smokes costs the employer an average of from 600 to 900 dollars/year. Last year, U.S. companies spent approximately 10 percent of their total operating budgets on health care costs. To help companies contain health-related expenditures, the factors necessary for an effective cost-conscious health promotion program are outlined. These include commitment from top corporate levels, a supportive environment for employees' health behavior change, knowledge of employee health values, acknowledgement of the value of employees' time, an emphasis on intervention instead of screening, the importance of experience-oriented learning, a systematic evaluation of program effectiveness, and the establishment of a health officer position. In addition to smoking cessation and reduction, the guidelines are also applicable to seat belt use, chemical dependency, exercise, diet and nutrition, stress, and hypertension management.

84–0615. Richmond, J. B. **Ending the Cigarette Pandemic.** *New York State Journal of Medicine* 83(13): 1259, December 1983.

The Federal Government's minimal budgetary commitments to counter the tobacco industry's multimillion dollar campaigns against advertising and smoking legislation indicate that the medical profession must take the initiative and address the smoking issue as a number one health priority. Future efforts to end the cigarette pandemic must emphasize prevention in six major areas: (1) victimization of women (lung cancer deaths increased from 4.6/100,000 women in 1950 to 20.9/100,000 in 1982, while, of 1983 cancer deaths among females, 17 percent will be due to lung cancer and 18 percent to breast cancer); (2) adolescents (75 percent of smokers are dependent on cigarettes by age 20); (3) smoking cessation strategies (over 90 percent of smokers wish to quit, and it is the medical profession's responsibility to reinforce this choice); (4) nonsmokers' rights to clean indoor air (especially children's); (5) low-tar, low-nicotine misconceptions (there is no safe cigarette); and (6) disclosure of chemicals and artificial tobacco substitutes added to low-yield cigarettes to increase palatability. It is indicated that the tobacco industry is attempting to portray smoking as a civil rights issue, since the reliability of health data cannot be doubted.

84–0616. Schinke, S. P.; Gilchrist, L. D. **Primary Prevention of Tobacco Smoking.** *Journal of School Health* 53(7): 416-419, September 1983.

Data show that primary prevention groups improved sixth graders' problem solving, decisionmaking, and interpersonal communication regarding tobacco use. Sixth grade students (30 girls and 26 boys, mean age 11.70 years) were randomly assigned to one of four experimental conditions: pretest and primary prevention; primary prevention; pretest; and neither pretest nor primary prevention. In eight group sessions, primary prevention students learned smoking prevention-oriented facts, problem solving, decisionmaking, self-instructions, and interpersonal skills. All students completed posttests and a 6-month followup. Compared with untrained controls, trained students had greater knowledge of smoking, greater perspective on tobacco problems, better linkage between problems and solutions, more insight on the consequences of nonsmoking decisions, and greater nonverbal and verbal competence in tobacco use situations. Followup data associated trained students with stronger commitments to tobacco abstinence, more frequent refusals of tobacco, and less smoking than controls. Midway through seventh grade, 8 percent of trained students and 37.5 percent of controls had smoked in the most recent month. (Auth. Abs. Mod.)

84–0617. Snow, B. R. **Workplace Strategies for Smoking Cessation.** In: *Progress in Cancer Control. III: A Regional Approach.* Progress in Clinical and Biological Research, Volume 121, New York, Alan R. Liss, Inc., 1983, pp. 113-114.

A worksite smoking cessation program currently under development by the Cancer Control Division of Columbia University emphasizes the psychological, physical, and environmental factors that promote and reinforce the smoking habit. Topics include (1) health education on the synergistic effects of smoking and occupational exposure to chemicals in the development of cancer; (2) use of coworker groups for discussion; (3) analysis of environmentally produced stress; and (4) mutual support as an important factor in social context of smoking cessation. This program will also include training of workers to conduct future smoking cessation programs at the worksite.

84–0618. Stachnik, T.; Stoffelmayr, B. **Worksite Smoking Cessation Programs: A Potential for National Impact.** *American Journal of Public Health* 73(12): 1395-1396, December 1983.

Data from three pilot studies of small worksite smoking cessation programs demonstrate that programs that appeal to all smokers in an organization, not just those who have stated an interest in achieving cessation, will attract a high percentage of smokers and assist an unusually high number to achieve and maintain abstinence. Programs were conducted at (1) a bank's headquarters office, (2) an organization providing services to hospitals, and (3) the offices of a manufacturing company. The percentage of smokers enrolled in the programs were 70, 47, and 54, respectively. The 7-month program, which included monetary incentives and contracts, was based primarily on weekly group meetings for the first 3 months, every other week during months 4 and 5, and every third week thereafter. The program described may be unnecessarily complex for implementation on a large scale, but there is evidence that financial incentives alone can have a significant impact. While the cost might reach the minimum benefit estimate, program costs are for one time only, while company savings accrue each year the smoker remains abstinent. Abstinence rates at 6 months ranged from 81 to 95 percent in the pilot studies. (Auth. Abs. Mod.)

84–0619. Strecher, V. J. **A Minimal-Contact Smoking Cessation Program in a Health Care Setting.** *Public Health Reports* 98(5): 497-502, September-October 1983.

In a minimal-contact smoking cessation program at the Ann Arbor, Michigan, Veterans Administration Hospital, patients under care participate in a brief consultation about their smoking with a practitioner. For inpatients, the consultation occurs near the time of discharge from the hospital. Toward the end of the consultation, if the patient decides to try to quit smoking, he or she is given a self-help smoking cessation kit in a 3-week diary format. With the practitioner, the smoker fills out the first series of exercises in the kit, including a smoking awareness test, and signs a stop-smoking contract that is cosigned by the practitioner. The patient takes the kit home, where the remainder of the kit's instructions and exercises are to be followed. Compliance is encouraged by weekly telephone calls from the health practitioner to check on the patient's progress and by offering the patient a State lottery ticket for each week the diary is completed. (Auth. Abs. Mod.)

84–0620. Swartz, J. **Winnipeg Conference Declares International War on Smoking.** *Canadian Medical Association Journal* 129(6): 636-641, September 15, 1983.

War was declared not on people who smoke, but on the transnational companies that manufacture and promote the smoking epidemic. The epidemic is waning in industrialized countries, where smokers are now a minority, but in Third World nations, smoking is increasing so rapidly that it is estimated that in the next two decades, tobacco will be the leading cause of death. Besides the health problems, tobacco exerts the single heaviest demand on the Third World's wood supply, usurps large proportions of incomes, and occupies land that could be used for crops. These countries are called on to prohibit tobacco promotion, tax such products heavily, prevent sales to children, provide health warnings, and regulate cigarette yields. Changing public attitudes and legislation concerning passive smoking, and the growing number of antismoking activist groups are discussed. The roles of nicotine gum, quit programs, physician encouragement, social support of nonsmoking, and low-yield products for those who can't quit in curbing the epidemic are described. It is concluded that smoking is a public health catastrophe, and 90 percent of the cure is political action.

84–0621. Vartiainen, E.; Pallonen, U.; McAlister, A.; Koskela, K.; Puska, P. **Effect of Two Years of Educational Intervention on Adolescent Smoking (The North Karelia Youth Project).** *Bulletin of the World Health Organization* 61(3): 529-532, 1983.

Results of a 2-year smoking intervention program among 13- to 15-year-olds showed that there were significantly fewer smokers among the intensive intervention school students (conducted by project workers) and the teacher-conducted intervention students compared with reference school students at second followup. The program, conducted in the county of North Karelia in Finland, attempted to teach students skills to resist the social pressures to smoke. Older students (peer leaders) were trained to conduct the program, which was directly managed in two schools and voluntarily implemented by the staff of about half of the remaining 23 schools in the county. Both types of intervention were associated with a significant reduction in self-reported onset rates of smoking. At followup, 17 percent of the intervention students smoked daily compared with 24 percent of the reference school students ($p<0.05$). The overall change in the percentage of smokers was +15 in the intensive intervention, +11 in the less concentrated program, and +36 in the reference area. Among girls, percentages of change were +15, +21, and +23, respectively. (Auth. Abs. Mod.)

See also, **84–0572**

SMOKING CESSATION METHODS

84–0622. Astrup, C. **Nicotine and Behavior Therapy of Heavy Smokers. (Letter).** *Pavlovian Journal of Biological Science* 18(2): 113, April-June 1983.

The smoking cessation treatment described involves a gradual reduction in cigarette consumption supplemented with nicotine chewing gum. Subjects receive a nine-page description of the treatment and are instructed to record their consumption for 3 days. During the first month, consumption is reduced to 5 cigarettes/day. Free access to nicotine is allowed for 2 months. The third month is used to eliminate chewing the nicotine gum. Since 1974, about 100 persons have received the treatment.

84–0623. Battista, R. N. **Adult Cancer Prevention in Primary Care: Patterns of Practice in Quebec.** *American Journal of Public Health* 73(9): 1036-1039, September 1983.

Among 430 general practitioners surveyed in Quebec in late 1981 and early 1982, 98 percent reported giving cessation of smoking support to patients. Methods used included providing information, Nicorette (nicotine) gum, group therapy, acupuncture, hypnotism, tranquilizers, and threatening to terminate the medical relationship. With asymptomatic patients at high risk of developing lung cancer, 77 percent of physicians reported using X-rays as an early detection procedure; 66 percent used X-rays yearly or more often. General practice measures for detection and prevention of breast, cervical, and colorectal cancer are also presented. (Auth. Abs. Mod.)

84–0624. Griffith, E. E.; Crossman, E. **Biofeedback: A Possible Substitute for Smoking, Experiment I.** *Addictive Behaviors* 8(3): 277-285, 1983.

Two smokers who learned to produce high levels of 8 to 12 Hz occipital EEG activity without biofeedback equipment were able to quit smoking. Biofeedback techniques were used to identify physiological variables that contribute to maintenance of cigarette smoking, and to investigate the hypothesis that smoking frequency would decrease when individuals were trained via biofeedback procedures to increase 8-12 Hz occipital EEG activity as a substitute for smoking. Results from monitoring six male smokers as they smoked one cigarette showed that (1) four smokers increased the percent of time they were producing 4 to 8 Hz brain waves; (2) five smokers increased their heart rate; and (3) six smokers decreased their 8 to 12 Hz activity. Immediately after consuming the cigarette, all subjects demonstrated an increase in heart rate and four demonstrated a decrease in skin temperature. There did not appear to be any specific consistent brain wave change across subjects. The two subjects who quit were still abstinent at 8-month followup. (Auth. Abs. Mod.)

84–0625. Hataway, J.; Hughes, G. **Physicians and the Patient Who Smokes: Motivational Aspects.** *Alabama Medicine* 53(5): 26-28, November 1983.

Physicians have produced, through personal counseling, 1-year smoking cessation rates ranging from 30 to 60 percent, and from 40 to 60 percent in patients with chronic heart and lung disease. A thiocyanate-validated smoking cessation rate of 46 percent at 6 years was achieved through the Multiple Risk Factor Intervention Trial. Even when patients state that they do not wish to quit, physicians have the responsibility to use clinical judgment to assess actual motivation, ask smokers to consider quitting, and inform patients of smoking hazards. A physician's office is an excellent place to conduct smoking cessation counseling due to (1) the physician's role as a healer; (2) the patient's position as "captive audience"; and (3) repeated documentation that long-term results are impressive. Frequent visits and a combined factual and behavioral intervention approach are recommended. Physicians are advised to allow as much time per visit for smoking cessation counseling as they would for other office visits, and to list cigarette smoking as a prominent medical problem on the patient's office and/or hospital chart. Smoking cessation materials from the American Lung Association, American Cancer Society, or American Heart Association should be on hand and actively utilized. A project to improve smoking cessation efforts in the physician's office is discussed.

84–0626. Hill, D. **The Family Physician's Influence on Patients' Smoking Habits.** *Australian Family Physician* 12(9): 678-679, September 1983.

While a physician's counseling and leaflets combined with followup achieved a 23 percent smoking cessation rate (compared with 12 percent for advice and leaflets only), the 5 percent success rate achieved in a nonselected group with only 2 minutes of a doctor's time is also valuable. Increasing support for the physician's role in smoking cessation is described: preliminary results of a medical school program in New South Wales are promising; British organizations have provided all general practitioners with smoking cessation materials and guidelines, coordinated with a BBC television series; and antismoking media campaigns are increasing in Australia, enhancing physicians' effectiveness. A nicotine chewing gum (Nicorette) appears to be an effective smoking cessation aid; 21 percent of patients chewing placebo gum quit smoking over a 12-month period, compared with 47 percent of Nicorette users in a randomized, double-blind London trial. However, general acceptance of smoking as a problem to be cured by physicians (prescribing gum or drugs) could be deleterious to health education.

84–0627. Lotecka, L.; MacWhinney, M. **Enhancing Decision Behavior in High School "Smokers".** *International Journal of the Addictions* 18(4): 479-490, 1983.

Participants in the cognitive-behavioral group (n=23), but not in the information only group (n=26), achieved significant smoking reduction at both 1-month and 3-month posttests. While further research is planned to clarify the most significant components of the program, analysis indicates that enhancing participants' belief in and use of their decisionmaking power contributed to results. In the first stage of the study, personal control beliefs of smokers (n=138) and nonsmokers (n=111) in six high schools were analyzed. This attitudinal information was used to embed implicit suggestions into an interventional format. The cognitive-behavioral intervention in two high schools involved giving identified smokers various reinforcements for coping behaviors relevant to cigarette control. Matched groups in two other high schools received popular tips on how to quit smoking. Subjects receiving the cognitive-behavioral program revised their ideal level of marihuana consumption downward and differed on certain other beliefs, compared with the popular tips group. Motivational and self-regulative characteristics of participants were also examined through in-depth questioning. (Auth. Abs. Mod.)

84–0628. Prue, D. M.; Davis, C. J.; Martin, J. E.; Moss, R. A. **An Investigation of a Minimal Contact Brand Fading Program for Smoking Treatment.** *Addictive Behaviors* 8(3): 307-310, 1983.

A self-help program, using a written manual and limited duration telephone calls (to add structure to the treatment program) showed promise in the treatment of chronic smokers.

Of subjects (mean age 45.25 years) in a Veterans Administration smoking program, 85 percent had smoking-associated health problems, primarily cardiovascular (23 percent) and pulmonary (63 percent). On the average, subjects had attempted abstinence 4.8 times, consumed 32 cigarettes/day, and had smoked 41.5 pack-years. Two groups participated: the minimal contact brand-fading group (MCBF, n=20); and the control group (n=10), who had been told they would be put on a waiting list for treatment. In phase I, a lower nicotine brand was substituted every 2 weeks; phase II was abstinence, using standard behavioral strategies. After 6 months, 17 percent of the MCBF group were abstinent, compared with 10 percent of controls. The control group then underwent treatment. After a year, 23 percent of the MCBF group was still abstinent.

84–0629. Schneider, N. G.; Jarvik, M. E.; Forsythe, A. B.; Read, L. L.; Elliott, M. L.; Schweiger, A. **Nicotine Gum in Smoking Cessation: A Placebo-Controlled, Double-Blind Trial.** *Addictive Behaviors* 8(3): 253-261, 1983.

Nicotine gum was significantly more effective than a placebo on cessation rates in a clinic support study. Subjects in the clinic support group (17 females, 13 males) and the placebo group (19 females, 11 males) chewed nicotine or placebo gum ad libitum. A survival analysis showed the two groups differed significantly in successful abstinence over time ($p<0.03$). Differences between groups appeared early (within weeks) and, at 6 months, mean success rates were 48 percent for nicotine gum and 20 percent for placebo. After 1 year, rates were 30 and 20 percent, respectively. In a pilot study where intervention was minimal, neither gum was effective in cessation. The enhanced short-term success rates with nicotine gum in the clinic study are attributed to an effective interaction between the active preparation and clinic support. Long-term cessation may require extended maintenance procedures or identification of optimal gum use. (Auth. Abs. Mod.)

84–0630. Zeitlin, D. I.; Dismuke, S. E.; Miller, S. T. **Antismoking Efforts by Physicians. (Letters).** *Journal of the American Medical Association* 250(23): 3161-3162, December 16, 1983.

A random survey (n=1,000) of Los Angeles County Medical Association members showed that 90 percent of 500 respondents (9 percent smokers) felt a professional obligation to help patients give up smoking despite frustration with discouraging results and the need for information concerning the most effective methods. In the most effective strategy (30 percent success) among those described, the physician simply informed patients who smoked that he would not continue to take care of them if they continued to smoke. The authors of a previous commentary regarding physicians' health promotion responsibilities support an optimistic viewpoint of health promotion, emphasizing the need for ongoing education of physicians and the continuation of antismoking efforts. While behavior change is based on a continuum (i.e., multiple experiences), of which physicians comprise only one link, it is stated that physicians must do their appropriate share.

See also, **84–0577**

TOBACCO PRODUCT ADDITIVES

84–0631. Boden, R. M. **Use of Prins and Derivatives Thereof in Augmenting or Enhancing the Aroma or Taste of a Smoking Tobacco Composition or Smoking Tobacco Article Component.** United States Patent No. 4,413,639, November 8, 1983, 38 pp.

A process for augmenting or enhancing the aroma or taste of a smoking tobacco composition is described, which provides an organoleptically improved product having specific Oriental, fruity, rum-like and Turkish tobacco-like notes prior to and on smoking. The additives' nuances may be readily maintained and controlled regardless of the tobacco components of the blend. To produce the desired flavors, an additive containing at least one Prins reaction product or Prins reaction product derivative is prepared in accordance with the described process. Other flavoring and aroma enhancers may be added, separately or in admixture, to the smoking tobacco material, the filter, or the leaf or paper wrapper. The proportions may be varied according to taste, but satisfactory results are obtained if the proportion by weight of the sum total of one or more Prins reaction products or Prins reaction product derivatives produced to smoking tobacco material is between 250 ppm and 1,500 ppm of the active ingredients to the smoking tobacco material. Any convenient method of application may be used, and only a portion of tobacco needs to be treated before blending with other tobaccos. (Auth. Abs. Mod.)

84–0632. Scheideler, G. **Components for Basic Tobacco Flavor.** *Tobacco International* 185(19): 14, 16, 18, September 16, 1983.

Natural tobacco flavoring agents (TFAs) introduced by Haarman and Reimer include Continental, Burley, and Virginia Continental; and Continental, Burley, Virginia, and Special Blend for filters. TFAs (designed to improve overall tobacco flavor and increase smoker satisfaction) are currently registered as tobacco flavor concentrates, and are 10 times the strength of normally available tobacco extracts. Dosages of the three basic TFAs range from 0.01 to 0.03 percent, depending on tobacco blend quality. Impressive effects were observed at the 0.03 percent level with reconstituted tobaccos. Sensory evaluations show that certain TFA characteristics are consistent: boosting effect is particularly and significantly associated with the low- and ultra-low-tar segments, especially when using TFA for filters; smoothing effect occurs particularly with normal and full-flavored cigarettes, significantly with cigarettes using high portions of reconstituted tobacco and expanded ribs; balancing effect is observed through all segments; mouth-watering effect is significantly high in low- and ultra-low-tar cigarettes, especially with the four white filter TFAs; and the altering effect is observed only when the basic brown TFAs are used at incorrect levels.

TOBACCO MANUFACTURING AND PROCESSING

84–0633. Dwyer, R. W., Jr.; Fleming, M. L. **Cigarette Mouthpiece.** United States Patent No. 4,413,641, November 8, 1983, 6 pp.

A mouthpiece is described that is attached at one end either to the mouth end of a filter of a cigarette, or the filter of a filtered cigarette holder. A channel through the mouthpiece is of substantially the same area at the inlet end as the filter, but from the inlet end to a point between that end and the outlet, the transverse sectional area continuously decreases, and from that point to the orifice is either the same or continuously increases until it is the same transverse sectional area as the orifice. Smoke passes through the filter and into the inlet end of the mouthpiece, exiting at the outlet end in a preselected narrow pattern. The mouthpiece improves the subjective perception of cigarettes that have a low "tar" delivery and enhances the subjective effect of the smoke, which may have been attenuated by dilution or filtration.

84–0634. Floyd, T. S.; Rhoden, D. G.; Bradley, W. E. **Ventilated Thermoplastic Polymer Foam Filter Rods.** United States Patent No. 4,411,280, October 25, 1983, 13 pp.

Ventilated thermoplastic polymer foam cigarette filter rods and processes for their preparation are described, which provide virtually unlimited tip design flexibility and eliminate costly ventilating procedures. The ventilated thermoplastic polymer foam filter rods are characterized by the presence of at least some cells which are bridged by fibrils, the rod being surrounded by a high density skin structure having a discontinuity such as to produce a ventilation of at least 50 percent. Discontinuity in the skin structure may be produced by skin solvent etching, skin abrading, skin perforating, and skin cutting. (Auth. Abs. Mod.)

84–0635. Greig, C. C.; Hook, R. G. **Smoking-Material Rods and a Method of Making Such Rods.** United States Patent No. 4,416,295, November 22, 1983, 6 pp.

A rod of smoking material, for use as a smoking article or part thereof, is described. The rod is formed by many laminiform self-sustaining smoking-material elements, for example discs, extending transversely along the rod and in face-to-face contact. The thickness of the elements (between 0.1 and 0.8 mm) may vary within the rod or from element to element along the rod. The smoking-material elements may be formed of tobacco leaf lamina, reconstituted tobacco sheet, tobacco substitute in sheet form, or of any other sheet form material suitable for inclusion in the smoking-material rod of a smoking article. The material of the elements may have inherent smoke-permeability sufficient to afford an acceptable pressure drop through the rod, or the elements may be formed or provided with smoke passages therethrough, with either the same relative distribution of passages from one element to the next or with adjacent elements so oriented in relation to each other as to provide smoke passages of predetermined form through the rod. The smoking material of the elements preferably extends over an area less than the cross-sectional area of the rod, suitably over 10 to 40 percent of that area. One or more additives may be applied to some or all of the elements, either uniformly or in selected parts thereof. The elements may be secured together by adhesive or enclosed in a wrapper, and may be formed with protrusions from or undulations of faces thereof. (Auth. Abs. Mod.)

84–0636. Moragrega, M. S. **Impact Filter for Tipped Cigarettes.** United States Patent No. 4,414,989, November 15, 1983, 4 pp.

An impact filter for tipped cigarettes is described that is effective in retaining noxious ingredients in cigarette smoke (preferably tar, an agent that is considered to be highly carcinogenic). The filter is comprised of a cylindrical chamber, of a somewhat hard and transparent material, with a diameter equal to that of the cigarette, coupled to a lower base containing a series of very small peripheral orifices (perpendicular to smoke flow) that impart greater velocity to the smoke. On impact with the inner surface of the upper base, the smoke is rejected and a large portion of the tar is deposited on both bases, the smoke finally ascending through a downward tubular orifice in the center of the upper section. The chamber is preferably located between two conventional portions of absorbent material, conventionally wrapped, to give the appearance of a normal filter.

84–0637. New Scientist. **Cigarette Tar Tests Do Not Work.** *New Scientist* 99(1375): 749, September 15, 1983.

In a New York court, tobacco industry representatives admitted that there is no way of testing a cigarette for tar and nicotine content that can take into account the different habits of smokers. Thus the tar tables, which purport to grade the danger from cigarettes, have little meaning in the real world of smokers. The continuing controversy over the Barclay claim of "99 percent tar free" is reviewed, noting that the cigarette has been the subject of litigation in several European countries. If a smoker blocks the four aerodynamic holes in the filter tip, the Barclay could deliver 10 to 11 mg tar; the Federal Trade Commission rates it at 7 mg tar. In Holland, the government-financed Dutch Foundation on Smoking and Health blames an increase in smoking rates on the so-called health cigarette. The "science" behind the filter is explained.

84–0638. Sanford, R. A.; McMurtrie, A. **Cigarette Filter.** United States Patent No. 4,406,295, September 27, 1983, 13 pp.

A cigarette filter that reduces tar (mainly by ventilation), while also providing smoke filtration, is described. The filter includes a porous filter rod with a nonporous or air-impermeable wrapper, and ventilating air grooves, embedded in the wrapped filter rod, that extend from one end of the filter rod a preselected distance (generally longitudinally). An air-permeable tipping material circumscribes the wrapped filter rod, providing a path for ventilating airflow into the grooves. The wall of each grooves is permeable to air over virtually its entire length, or may be permeable to airflow only adjacent to the mouth end of the filter. Under certain smoking conditions, most (if not all) of the ventilating air will flow from the grooves into the smoker's mouth through the open end of the grooves at the mouth end of the filter rod. Under other smoking conditions, at least a part of the ventilating air will pass through the air-permeable walls, or portions thereof, of the grooves and into the filter rod, wherein it mingles with and dilutes the smoke. (Auth. Abs. Mod.)

84–0639. Selke, W. A. **Making the Cigarette Do Just What You Want It to Do.** *Tobacco International* 185(19): 8, 10, 12, September 16, 1983.

Achieving independent control of individual aspects of cigarette performance and smoke constituent interrelationships requires simultaneous manipulation of a growing array of variables. In addition to previous selections, which were limited to the tobacco and filter plug, choices now include cigarette paper (for permeability and burning rate); tipping paper (perforated) and plug wrap (porous) for ventilation; and reconstituted tobacco (to adjust particulate delivery, burning rate, nicotine delivery, carbon monoxide (CO) delivery, and nitrate content). Cigarette engineering techniques for (1) reducing CO while maintaining unchanged tar delivery and puff count; (2) increasing nicotine delivery without changing tar delivery; and (3) increasing puff count while keeping tar delivery unchanged are described, as are interactions and side effects of components.

84–0640. Yagi, M. **Tobacco Smoke Filter.** United States Patent No. 4,414,988, November 15, 1983, 10 pp.

A filter capable of effectively removing carcinogenic substances (benzo(*a*)pyrene and its derivatives) from tobacco smoke is described. The filter contains an aqueous solution of a compound having a metallic ion and especially ferric ion binding protoporphyrin ring structure, which does not impair the flavor of tobacco smoke when compared with conventional smoke filters. A porous carrier of any conventional filter material can be impregnated with the carcinogen removal agent, such as hemin, or the agent can be incorporated into a cigarette or pipe in any known manner, such as a rupturable capsule. The aqueous solution can remove the benzo(*a*)pyrene from tobacco smoke by using 0.1 to 10 mg, preferably 0.1 to 2 mg of at least one of the described compounds.

TOBACCO ECONOMICS

84–0641. Ahmad, M. **Spotlight on Pakistan: Struggling to Survive.** *Tobacco Reporter* 110(10): 24-26, 28, October 1983.

The inflated costs of raw materials, taxes, and antismoking propaganda have combined to limit anticipated growth of the Pakistan tobacco industry to only 3 percent for 1983. In mid-June, when the government raised the duty on most brands from 50 to 52 percent and reduced taxes from 72 to 70 percent on a few higher priced brands, manufacturers raised almost all brand prices. With annual per capita income equal to approximately 350 U.S. dollars, per capita tobacco consumption at 1 kg is one of the lowest rates worldwide. Only 55 percent is consumed in cigarettes; about 70 percent of the population live in rural areas where tobacco is used in noncigarette forms. A shift toward filter tips is reflected in a rise from 1981 production of 38 percent to 44.3 percent in 1982. Data are tabulated for 1982 cigarette production and market shares, top 20 brands 1982, 1982 exports by brand, and leaf requirements.

84–0642. Gabriel, L. **German Cigars: Profile of a Depressed Market.** *Tobacco Reporter* 110(11): 46, November 1983.

For the first 6 months of 1983, sales figures for German cigars and cigarillos (919 million pieces) showed an upward trend compared with the same period in 1982. Both price increases (attributed to tobacco product taxation) and resistance among younger smokers are considered obstacles to cigar and cigarillo sales. Currently, cigars represent 28 percent of the market; cigarillos 68 percent and rising; and cheroots 4 percent and falling. Exports are significant (220 to 250 million pieces annually), as are imports (yearly volume, 230 million pieces). Additional sales and price data are provided.

84–0643. Gabriel, L. **European Cigars: In Search of New Markets.** *Tobacco Reporter* 110(11): 44-45, November 1983.

While analysis of European Economic Community (EEC) cigar consumption trends indicates that it may be possible to identify and develop new markets, tax burdens must be lifted before stability can return. Meanwhile, with the exception of Italy, cigar consumption continues to decline. Positive previous market patterns include (1) soaring consumption of very small, less expensive cigars and cigarillos in the United Kingdom from 1960 to 1975; and (2) tripling of consumption in France from 1960 to 1980, as EEC cigar companies secured French market positions. Declining markets in West Germany, the Netherlands, Belgium, and Denmark, attributed primarily to price increases, are described, and cigar and cigarillo EEC consumption data are tabulated by year for 1960 through 1982.

84–0644. Humphries, B. **Shifts in U.S. Cigarette Industry Alter N. C. Economy.** *Tobacco International* 185(20): 42, September 30, 1983.

Changes in cigarette manufacturing have had a considerable economic impact in North Carolina. Only 1.7 pounds of tobacco are now required to make 1,000 cigarettes (40 percent less than the 2.7 pounds needed in 1950-1954). The reduction is due to (1) use of filters, which comprise 30 to 40 percent of a cigarette's length, on 93 percent of U.S. cigarettes; (2) development of homogenized leaf, which increases the yield of cigarettes/pound of tobacco; and (3) changes in price and quality relationships among various types of tobacco. Farmers have been especially hurt by the dramatic downward shift in the proportion of domestic flue-cured used in blended cigarettes (from 60 percent 30 years ago to 35 percent today). Also, excise taxes have reduced demand for U.S. tobacco by about 3 percent.

84–0645. Palacpac, O. S. **West Germany Again Top Buyer; Bill for "Tobacco Bank" Proposed.** *Tobacco International* 185(20): 6, 8-9, 12, 15, 19, September 30, 1983.

Statistics indicate some recovery in the Philippine tobacco industry over previous year losses, with increased exports more than balancing increased imports. The Federal Republic of Germany led 14 buyers in 1982, purchasing 4.67 million kilos, worth 11.08 million dollars (34.66 percent of total quantity of leaf tobacco fillers and binders sold). The United States ranked second, followed by Japan and the United Kingdom/Northern Ireland. Domestic prices for tobacco and tobacco products rose as a result of additional taxes imposed by the National assembly. Four bills affecting the industry are still pending: (1) creation of the Philippine Tobacco Bank to help in financing the tobacco program; (2) help for Virginia tobacco growers by allowing them sell to any trader; (3) coverage for tobacco farmers under Social Security; and (4) amendment of the tax law on Virginia-type cigarettes. Cigarette advertising has been heavy in major cities and a bill to ban all media advertising of tobacco products, if it becomes law, would result in an estimated loss of 5.4 million dollars to advertising companies. A governmental agency attempted to have local cigarette manufacturers include health warnings on cigarette packages, but this was not enforced.

84–0646. Shien, T. **In the Offing: Freer Import Access, Eclipse of JTS's Monopoly Status.** *Tobacco International* 185(18): 5-6, 8, 10, 12, 14, September 2, 1983.

Developments in Japan indicate that the issue of sales of imported cigarettes may be resolved within a few years, since the only remaining point of contention is the distribution channel into retail outlets, currently controlled by the Japanese Tobacco and Salt Public Corporation (JTS). The number of retail outlets allowed to sell imported brands was doubled in 1982 (from 20,000 to 40,000) and 30,000 more will be added in 1983. Beginning in 1984, there will be no restrictions on who may sell imported tobacco. Import duties have also been reduced, making imported brands more competitive, since prices on domestic brands have been increased. A committee recommends that a new company be set up to handle cigarette imports and distribution, and the JTS would, therefore, no longer be a monopoly. While protests are raised by tobacco growers' associations, the government is expected to follow the course to a freer tobacco market. Production figures for 1982 are also discussed and technological improvements in growing and processing tobacco (which have reduced tobacco use per unit and labor costs) are described. Total sales of cigarettes increased slightly in 1982 (0.9 percent), but estimates for 1983 are for only 98.6 percent of that total, while sales of imported brands in 1983 are expected to be 143.8 percent of the 1982 total.

84–0647. Stone, M. **Dutch Cigars: The Strategy: Aggressive Marketing.** *Tobacco Reporter* 110(11): 48, 50, 52, November 1983.

Signs of stabilization in world cigar consumption are now evident after recent years of decline. Possible further decrease in traditional markets (i.e., Germany and Holland) may be balanced by further market growth in the United Kingdom, France, Italy, and potential markets outside Europe. Worldwide cigar consumption totals 17,500 million pieces. Imports represent 15 percent of all cigars smoked; Holland, with 1,500 million pieces and 60 percent of the market, is the main exporter. The Henri Wintermans Company produces nearly 40 percent of Dutch (and more than 20 percent of worldwide) cigar exports. Wintermans' growth, brands, export data, and marketing strategies are summarized. Cigar consumption in million pieces and annual adult per capita consumption are tabulated for selected countries, as are export figures.

84–0648. Tobacco International. **Korea. Hail Damage Not Serious; Monopoly Urges Less MH, More Compost.** *Tobacco International* 185(21): 5-6, October 14, 1983.

Color, structure, leaf body, and average number of leaves/plant (18 to 21) are improved in 1983, compared with the 1982 harvest. Stimulated tobacco growth is attributed to a short rainy season and hot weather. For 1984, the Office of Monopoly, which controls Korean tobacco production, has (1) persuaded farmers to reduce use of maleic hydrazide (MH) to hold MH residue on tobacco to acceptable world levels; (2) decided to substitute MH for other sucker control chemicals; (3) encouraged farmers to use more than 12 tons of compost/ha before transplanting (to produce high-quality tobacco with low nicotine and tar); and (4) educated farmers concerning fertilizer use and high-topping. Korean leaf tobacco export procedures are described, and exporters are listed.

84–0649. Tobacco Reporter. **Spotlight on India: Taxes Hamper Cigarette Sales.** *Tobacco Reporter* 110(10): 32-34, October 1983.

While 1982 cigarette production rose 7.4 percent over 1981 levels (93.2 billion pieces, 29 percent filter tips) and averaged 7,600 million pieces/month through October 1982, a price increase due to withdrawal of duty exemptions and increased excise taxes led to a drop in November sales. Growers are expected to reduce planted acreage in the 1983-1984 crop season, reflecting reduced prices, market problems earlier this year, and increased closing stock levels at year-end. India's overall 1982-1983 tobacco production declined to about 500,000 metric tons, approximately 5 percent below last year's record level output, primarily attributed to adverse weather conditions affecting bidi tobacco. Reduced prices reflected predominance of lower and medium grades and declining export demand (related to reduced Chinese purchases). Data are tabulated for 1982-1983 supply and distribution, 1982 exports of manufactured products and leaf, and revenue receipts from tobacco excises for 1982-1983 and 1983-1984.

84–0650. Tobacco Reporter. **Smokeless Tobacco: The Bright Star of the US Market.** *Tobacco Reporter* 110(11): 68-69, November 1983.

During the first 6 months of 1983, moist snuff sales registered gains of 7.3 percent compared with the same period in 1982, while sales of most smokeless tobacco products showed some decline. The increase was great enough to maintain overall snuff market growth of 4.2 percent, although dry snuff sales, continuing a downward trend, dropped 4.4 percent. Meanwhile, sales of loose leaf chewing tobacco gained 0.4 percent, firm plug lost 8.4 percent, moist plug dropped 13.8 percent, and twist/roll tobaccos fell 3.7 percent. Combined overall sales of chewing tobacco declined by 1.6 percent for the 6-month period. The export picture for U.S. smokeless products is also uneven. During the first half of 1983, overseas sales of moist plug rose by 64.7 percent, loose leaf exports gained 33.3 percent, and firm plug lost 16 percent. Total foreign shipments of chewing tobacco increased by 12.8 percent, compared with the January to June period in 1982. Exports of moist snuff were 1.9 percent higher. Marketwide, output of smokeless tobacco products rose 0.9 percent, sales gained 0.3 percent, and exports increased 3.5 percent, a positive and encouraging picture in an otherwise stagnant U.S. tobacco industry. Promotion of the U.S. Tobacco Skoal brand is discussed; and tabulated data for U.S. smokeless tobacco output, domestic sales, and exports, January through June (1982 and 1983) are provided.

84–0651. Tobacco Reporter. **Spotlight on Canada: Cigarettes Show First Annual Decline.** *Tobacco Reporter* 110(11): 64-66, November 1983.

A 0.3 percent drop in 1982 cigarette sales has become a 4 percent decrease for the year ending in June 1983. Reports from Canada's Health and Welfare Department indicate that the percentage of regular adult cigarette smokers aged 15 and above has declined to around 34 percent; correspondingly, production is expected to be lower for both 1983 and 1984. Declining consumption is attributed to both antismoking activities and automatic cigarette tax increases; taxes currently comprise about 60 percent of retail price. In 1982, the Federal Government received an estimated 1.15 billion Canadian dollars from tobacco product sales, and the provinces collected an additional 1.1 billion. An increase of 16.5 percent (5.5 million kg) in sales of domestically produced fine cut tobacco for roll-your-own cigarettes offset the manufactured product loss. Tabulated data are provided for Federal cigarette tax rates, and rates by province and territory; production by region and type for 1982 and 1983; and flue-cured imports and exports, 1981 and 1982.

LEGISLATION

84–0652. Ball, K. **Bypassing Bans on Cigarette Advertising. (Letter).** *Lancet* 2(8353): 803, October 1, 1983.

Philip Morris has removed the cigarette packs from the large cowboy advertisements prominently visible in Khartoum, following the Sudanese government's ban on all cigarette advertising. However, the cigarette packs are being replaced by cigarette lighters, indicating that major tobacco industries are prepared to go to any lengths to press their products on unsuspecting populations. Philip Morris and British American Tobacco, by far the largest suppliers of cigarettes to developing countries, have much to answer for.

84–0653. Business Week. **A Cloud of Controversy Over Smoking at Work.** *Business Week* 2813 (Industrial Edition): 53-54, October 24, 1983.

Smoking in the workplace faces its first ballot test in San Francisco, where passage of Proposition P would permit a smoking ban in an office based on the objections of a single employee. The vote is the result of a referendum drive, financed largely by tobacco companies, to overturn a similar ordinance passed by the city's board of supervisors. Nationally, 31 States now have laws limiting smoking in public areas and five regulate smoking in the workplace. However, the Tobacco Institute stresses that State legislators, not voters, passed the laws, and that voters in seven States defeated initiatives that sought to restrict smoking in public areas. Many large employers in the San Francisco area either have or are developing policies limiting smoking, and have not taken a position on the proposition. Such policies are clearly worrying the 25 billion dollar tobacco industry.

84–0654. Lutschg, J. H. **Why Uncle Sam Is Still Smoking.** *New York State Journal of Medicine* 83(13): 1278-1279, December 1983.

Governmental failure to discourage smoking, despite predictions that 100,000 Americans will die from smoking-related lung cancer and 200,000 will die from additional cigarette-related diseases in 1984, is attributed to long-term tobacco industry ties. Formation of the Tobacco Institute (now headed by a former Congressman) in 1958 merely formalized the longstanding alliance between cigarette manufacturers, agricultural officers, and Congressmen from tobacco-growing areas. Furthermore, the tobacco industry represents a source of funds to Congressmen: over 100 members from States growing little or no tobacco and more than 100 current members have received funds related to the Tobacco People's Public Affairs Committee, while many have received honoraria for speaking at tobacco industry functions. During the Carter administration, the director of Philip Morris corporate relations served as campaign liaison with business in 1976, and a Philip Morris board member served as chairman of the President's Council on Physical Fitness and Sports. In 1982, the budget of the Office on Smoking and Health of the Department of Health and Human Services was cut from 2.6 to 1.9 million dollars, less than 1/600th of the estimated 1.3 billion dollars spent advertising cigarettes. The National Cancer Institute (NCI) has spent 40 million dollars to develop less hazardous cigarettes, but has neglected (1) to acknowledge failure in reducing cigarette-related cancers, and (2) to include lung cancer 5-year survival rates in an NCI promotional publication. Also, tobacco was not listed as one of 102 regulated carcinogens in the United States in 1981 (while data associated one-third of all malignancies with smoking); and warnings are not printed on snuff, cigars, or loose tobacco, despite the experimental carcinogenic potency of a single cigarette tar component, benzo(*a*)pyrene, which is 50,000 times greater than potency of saccharin. Nevertheless, the word "cancer" appears on diet drinks, and candy bars must list ingredients and additives.

84–0655. Wigle, D. T. **Forced Smoking.** *Canadian Journal of Public Health* 74(4): 231-232, July-August 1983.

The need for legislation to protect Canadian nonsmokers from passive exposure to sidestream tobacco smoke (more appropriately labeled "forced smoking") is discussed, noting that concentrations of most toxic chemicals are substantially higher in sidestream smoke than in mainstream smoke. The health hazards of forced smoking, which are particularly severe in the fetus, infants, and children, require intense educational efforts to make people aware of the dangers in the home, since it is doubtful if governments will pass legislation to protect children or nonsmoking spouses. However, protection in the workplace can and should be provided under existing codes covering dangerous substances. Federal and provincial "clean indoor air" acts are also needed for the protection of nonsmokers in public areas such as hospitals, homes for the aged, buses, airplanes, stores, and shopping malls.

GENERAL

84–0656. Blum, A. **Cigarette Smoking and Its Promotion: Editorials Are Not Enough.** *New York State Journal of Medicine* 83(13): 1245-1247, December 1983.

Physicians cannot assume that patients are fully aware of smoking hazards or that the smoking issue is moralistic or a matter of informed consent, since the tobacco industry has become our leading health educator through advertising costing 1.5 billion dollars/year. Of nearly 9,000 continuing medical education courses offered in the United States in 1983, not one was devoted to analyzing methods of treating the smoking problem, which has been identified as the single most preventable cause of poor health worldwide and America's leading form of drug dependence. The tobacco industry has successfully allayed health concerns and undermined educational efforts by associating cigarette brands with enjoyable and healthful activities and by reinforcing glorified images of smoking. The success of cigarette advertising can be measured through continued high sales among young people and continued complacency of editors and publishers who refuse to admit the connection between cigarette promotion and smoking's high economic and physical toll. While newspaper editorialists across America call for greater physician accountability, newspapers continue to advertise cigarettes. Physicians must choose whether to adopt the tobacco industry's concept of health and disease or to act on the basis of their own knowledge, and they must realize that solving the smoking pandemic requires considering smoking in a variety of contexts.

84–0657. Cain, W. S.; Leaderer, B. P.; Isseroff, R.; Berglund, L. G.; Huey, R. J.; Lipsitt, E. D.; Perlman, D. **Ventilation Requirements in Buildings—I. Control of Occupancy Odor and Tobacco Smoke Odor.** *Atmospheric Environment* 17(6): 1183-1197, 1983.

Smokers account for nearly 90 percent of the demand for ventilation, although they constitute only about one-third of the population. In an environmental chamber, psychophysical measurements of odor, supplemented with certain physical measurements, were taken to examine ventilation requirements during smoking and nonsmoking occupancy. The facility provided the means for comparing impressions of visitors (persons who inhaled air from the chamber only briefly) with impressions of occupants. In an array of 38 conditions of smoking occupancy, the ventilation deemed necessary to satisfy 75 percent of visitors under customary conditions of occupancy equaled 17.5*l*/s/person. For both smoking and nonsmoking conditions, a combination of high temperature and humidity exacerbated the odor problem. During smoking, carbon monoxide rarely reached dangerous levels, but suspended particulate matter often reached levels considered unacceptable outdoors. For nonsmoking occupancy, 47 combinations of temperature, humidity, ventilation rate, and occupancy density were examined. Odor level depended entirely on ventilation rate per person regardless of the number of persons in the chamber. The ventilation necessary to satisfy 75 percent of visitors equaled only about 4*l*/s/person. Occupants, however, were satisfied with far less. The results highlight the energy penalty incurred in ventilation for smoking occupancy. (Auth. Abs. Mod.)

84–0658. Cowan, G. O. **Influence of Exercise on High-Density Lipoproteins.** *American Journal of Cardiology* 52: 13B-16B, August 22, 1983.

Significant increases in high density lipoprotein (HDL) and HDL_2 were found in nonsmokers (n = 19) and in men who had quit or reduced consumption (n = 15), but not in those who continued to smoke (n = 6). The effects of intensive rehabilitation on plasma HDL levels were determined in 40 men (aged 29 to 56 years) with ischemic heart disease. Exercise consisted of aerobic activities that induced up to 80 percent of the maximal heart rate during three 20-minute periods/day, 5 days/week, for 3 weeks. Data indicate that men with coronary artery disease may exercise vigorously, and exercise has a significant effect on HDL and HDL_2 levels, which correlate strongly with the incidence, severity, and mortality from coronary heart disease. Continued cigarette smoking, however, virtually negates any changes, while changes in those who quit are equal to results seen in nonsmokers.

84–0659. Daube, M. **Open Letter to . . . John Patten, MP, Under Secretary of State, Department of Health and Social Security.** *British Medical Journal* 287(6401): 1289-1291, October 29, 1983.

The realities of the job undertaken by Mr. Patten are outlined, noting that his predecessor was moved to the Department of the Environment, no doubt due to tobacco industry pressures that he was fighting with such determination. The position of Under Secretary has become identified with preventive medicine, and continuing efforts by someone not convinced by arguments of the tobacco industry and other supporters would include (1) a ban on all advertising and promotion of tobacco products; (2) tough and accurate health warnings (rather than the insipid nonsense offered by the cigarette manufacturers) on all cigarette packets; and (3) proper funding for health education. Other important targets would be regular yearly tax increases and upper limits on tar, nicotine, and carbon monoxide. The new minister is urged to act on the evidence that smoking kills about four times as many people as the total killed by alcohol, drugs, murder, suicide, road accidents, rail accidents, air accidents, poisoning, drowning, fires, falls, snakes, lightning, and every other known cause of accidental death combined.

84–0660. Forbes, W. F.; Thompson, M. E. **Estimating Economic Benefits and Losses Associated With Cigarette Smoking.** World Health Organization, Collaborating Centre for Reference on the Assessment of Smoking Habits, Faculty of Mathematics, University of Waterloo, Ontario, WHO/SMO/83.3, 1983, 28 pp.

General methodology for estimating smoking-related economic benefits and losses, and various difficulties associated with analysis, are summarized. Health care costs of smokers (and affected humans, particularly infants and children) are estimated for six age groups of the 1980 Canadian population. Per capita health care costs attributable to smoking are 15 percent for males and 10 percent for females. While two of three chapters specifically pertain to Canada, outlined methods of analysis are generally applicable to populations of other countries. Two main difficulties associated with a comprehensive risk— or cost—benefit analysis of cigarette smoking are (1) identifying an appropriate point of view (i.e., the individual, family, or society); and (2) estimating for each economic consequence the portion in some unit of measurement that is smoking-related. Furthermore, loss of human life, the greatest smoking loss that must be considered, is almost impossible to translate into monetary terms. See also 83-1729, 83-2068.

84–0661. Frecker, R. C.; Ashley, M. J. **Smoking and Health: A Survey of Research in Ontario.** *Canadian Journal of Public Health* 74(5): 343-347, September-October 1983.

The estimated 500 million dollars in tax revenue collected on tobacco sold in Ontario in 1980 is compared with a support base of 2,741,841 dollars from 40 funding sources (47 individual grants, 20 in-house budget allocations, 3 demonstration projects, 5 others) for 50 of 75 "current" smoking and health research projects in Ontario. Funding, distribution, appropriateness, and balance are discussed. A 1981 questionnaire survey showed that 70 investigators (representing 30.7 full-time equivalent researchers) spent an average 16.6 percent of their workweeks on smoking research (representing a total of 12.7 full-time equivalent smoking researchers active in Ontario). Investigations were currently under way at six Ontario universities (66 percent), three levels of government (23 percent), and various institutions (11 percent). Twenty projects were completed during the time frame surveyed (1978 to 1981), 44 were active in January 1981, and 11 were in the planning stages. (Auth. Abs. Mod.)

84–0662. Grant, R. W. **Cigarettes and Firesafety.** *Fire Journal* 77(4): 3, July 1983.

Support for the development and marketing of a firesafe (self-extinguishing) cigarette is expressed by the president of the National Fire Protection Association, noting that one-third of all fire deaths in the United States are due to fires started by cigarettes. While agreeing with the intent of the Cigarette Safety Act (H.R. 1880), it is suggested that the National Bureau of Standard's Center for Fire Research is best qualified to conduct the necessary testing and to develop criteria for firesafe cigarettes. However, standardization should be left to the private sector. The development of the firesafe cigarette is critical for preventing fire deaths, but public education is still needed. The Association is committed to both efforts to prevent the 2,200 deaths/year from smoking-related fires.

84–0663. Guidotti, T. L. **Adaptation of the Lifetime Health Monitoring Concept to Defined Employee Groups Not at Exceptional Risk.** *Journal of Occupational Medicine* 25(10): 731-736, October 1983.

Tobacco use history is one of many factors in the lifetime health monitoring program (LHMP) that can be used to tailor the program to individual participants. While the standard format includes smoking information every 5 years, increased smoking would indicate the need for special surveillance and cessation counseling. The LHMP described would replace the annual physical examination of employees with a plan modified for a particular population at risk. The initial target population studied consisted of managers whose risk profile is different from other populations but not specific to a particular exposure or unique hazard. Managers, especially women, tend to smoke more heavily than other white-collar workers, consume more coffee, and use medication such as aspirin and sleeping pills more frequently, and thus more closely resemble blue-collar workers. The program, now being used by a major employer in San Diego, is designed to create opportunities to provide educational and preventive services, and is less costly than other health examination programs. It is noted, however, that a prospective evaluation of the LHMP may be impractical.

84–0664. Klausen, K.; Andersen, C.; Nandrup, S. **Acute Effects of Cigarette Smoking and Inhalation of Carbon Monoxide During Maximal Exercise.** *European Journal of Applied Physiology* 51(3): 371-379, September 1983.

In 16 male smokers (5 to 15 cigarettes/day) a significant decrease in maximal heart rate (HR), and an increase in HR at rest was demonstrated during smoking but not while inhaling carbon monoxide (CO). The peak lactate concentration following maximal exercise was also significantly decreased after smoking. The acute effect of inhaling the smoke of three cigarettes was compared with the effect of inhalation of an amount of CO giving the same CO saturation of the arterial blood as smoking during rest and during maximal exercise on a Krogh cycle ergometer. Subjects (average age 24.5 years) were tested in the morning (1) after about 8 hours without smoking (control), (2) after inhalation of the smoke of three cigarettes, and (3) after CO inhalation. It was found that the average maximal rate of O_2-uptake (V_{O_2}max) decreased after both smoking and CO by about 7 percent. Endurance time at V_{O_2}max decreased 20 percent after smoking but only 10 percent during CO. Results suggest that the decrease in V_{O_2}max after smoking is due to the CO saturation of the blood, and hence to a decrease in the oxygen capacity of the blood, while the decrease in endurance time after smoking is a combined effect of the CO saturation and an increased cost of breathing caused by the smoke particles. It is further suggested that nicotine, or possibly some other components of the smoke, have an enhancing effect on the heart at rest, while an inhibition is seen during maximal exercise. Finally, it was found that the subjects who had a V_{02}max above the average for all subjects investigated were less susceptible to the effects of smoking than subjects with a V_{02}max below the average. (Auth. Abs. Mod.)

84–0665. New York State Journal of Medicine. **Confronting America's Most Costly Health Problem. A Dialogue With Surgeon General Koop.** *New York State Journal of Medicine* 83(13): 1260-1263, December 1983.

A 2-hour dialog between the Surgeon General of the United States Public Health Service, C. Everett Koop, M.D., and the editor of the New York State Journal of Medicine, Alan Blum, M.D., focuses on concerns related to cigarettes, which, although identified as society's most harmful product, are pervasively and insidiously associated with advertising images of glamour and "macho." Substantiated dangers of passive smoking to all nonsmokers are emphasized. Inequities of tobacco industry-approved warnings on labels and billboards are discussed, as are financial frustrations (industry finances allotted for lobbying in Washington, D.C., are five times greater than the budget of the Office on Smoking and Health). The major target of the 1.5 billion dollar cigarette advertising campaign (the most money spent on any U.S. product) is 14- to 16-year-olds, despite tobacco industry claims that ads are merely attempting to retain adult smokers. Limitations imposed on government (i.e., funding and freedom to speak without fear of consequences) indicate that the private sector (child-oriented industries, insurance companies, health charities) must energize public reaction to the smoking issue. Also discussed are health costs of smoking (13 billion dollars in 1979 plus 23 billion dollars work loss) vs. possible economic benefits; chemical additives that prevent cigarettes from self-extinguishing, causing 2,000 burn deaths annually in the United States; and the need to improve primary prevention of disease vs. current emphasis on reparative and rehabilitative health care.

84–0666. Solomon, L. C. **The Other Side of the Smoking Worker Controversy.** *Personnel Administrator* 28(3): 72-73, 101, March 1983.

Previous claims of increased costs due to smoking in the workplace are disputed, citing alternative methods of data interpretation and/or different or additional factors that could affect statistics. Cost issues discussed include insurance premiums, disability payments, medical costs, maintenance charges, and furniture replacement/property damage. Absenteeism is discussed in terms of occupational and demographic characteristics, and sick leave vs. vacation time. It is indicated that inefficiencies result when a special subgroup (i.e., smokers) is excluded from hiring consideration for reasons unrelated to job performance.

84–0667. Truempy, H. **Volkskundliche Uberlegungen zum Rauchen. [Smoking in History.]** *Therapeutische Umschau Revue Therapeutique* 40(2): 165-168, February 1983, German.

According to Swiss documents, cigarette use initially met with high resistance. The habit was imported from America and propagated mainly among soldiers, eventually becoming tolerated. Up to the middle of the 19th century, outdoor smoking was disapproved of, and women were forbidden to smoke cigarettes prior to 1918. Since 1945, smoking has increased among both men and women. Medical warnings became prevalent only after 1970.

AGRICULTURE

84–0668. Elkins, P. D.; Sheets, T. J.; Leidy, R. B. **Transfer of Disulfoton and Three Metabolites to Mainstream Cigarette Smoke.** *Tobacco International* 185(24): 51-54, November 25, 1983.

Disulfoton sulfone was the only metabolite of disulfoton found in cigarettes made from field-treated tobacco that contained <0.02, 0.03, 0.24, and 1.19 ppm of disulfoton sulfone for respective field treatment levels of the formulation equivalent to 0.0, 3.4, 6.7, and 13.4 kg/ha ai of disulfoton, an insecticide. The butts remaining after the cigarettes were smoked contained <0.04, 0.04, 0.23, and 1.03 ppm of disulfoton sulfone for the same treatment levels, indicating that none of the metabolite from the burning cone accumulated in the butts. Transfer rates in control cigarettes spiked at 5, 10, and 20 μg/cigarette averaged 14.9 percent for disulfoton, 8.1 percent for disulfoton oxygen analog, 0.4 percent for disulfoton oxygen analog sulfone, and 1.5 percent for disulfoton sulfone. Analyses again showed no accumulation of the four compounds in the butts. Filter tips reduced transfers of analytes to mainstream smoke by 59 percent for disulfoton, 32 percent for disulfoton oxygen analog, and 41 percent for disulfoton oxygen analog sulfone. There was no apparent effect of the filter on the transfer rate of disulfoton sulfone. No conversions of one analyte to another during smoking were observed. (Auth. Abs.)

84–0669. Ravishankar, G. A.; Mehta, A. R. **Regulation of Nicotine Biogenesis. 3. Biochemical Basis of Increased Nicotine Biogenesis by Urea in Tissue Cultures of Tobacco.** *Canadian Journal of Botany* 60(11): 2371-2374, November 1982.

Floral bud callus of *Nicotiana tabacum* L. var. Anand-2 were grown with urea as sole nitrogen source. Urea-treated tissues showed elevated nicotine content over the control. Activities of phenylalanine aminotransferase and tyrosine aminotransferase did not show close correspondence with growth or nicotine synthesis, whereas ornithine aminotransferase (OAT) activity exhibited correlation with nicotine biogenesis. Urea-treated callus showed higher OAT activity than the control. The activity of ornithine transcarbamylase (OTC) showed inverse correlation with nicotine synthesis. OTC and arginase activities were suppressed in urea-treated tissues. The implication of the results is discussed. (Auth. Abs.)

CHEMISTRY

84–0670. Adams, J. D.; LaVoie, E. J.; Shigematsu, A.; Owens, P.; Hoffmann, D. **Quinoline and Methylquinolines in Cigarette Smoke: Comparative Data and the Effect of Filtration.** *Journal of Analytical Toxicology* 7(6): 293-296, November-December 1983.

A refined method for quantitative determination of quinoline, isoquinoline, and each of the methylquinoline isomers in cigarette smoke was applied to the smoke of several different commercial cigarettes with and without filter tips. Quantitative analyses by capillary gas chromatography showed that quinoline, isoquinoline, and all seven methylquinolines were present in cigarette smoke. When cigarettes were analyzed with and without filters, the compounds were reduced by filtration, but not to the extent that tar was reduced. The relative composition of the methylquinoline fraction varied greatly among the brands studied, possibly due to differences in tobacco blends or to differences in filter construction. The significance of the data is discussed in view of the biological activity of specific isomers of these environmental pollutants. (Auth. Abs. Mod.)

84–0671. Alexander, A. J.; Goggin, P. L.; Cooke, M. **A Fourier-Transform Infrared Spectrometric Study of the Pyrosynthesis of Nickel Tetracarbonyl and Iron Pentacarbonyl by Combustion of Tobacco.** *Analytica Chimica Acta* 151(1): 1-12, July 1, 1983.

Fourier-transform infrared spectrometry was used to assess the concentration of nickel tetracarbonyl ($Ni(CO)_4$) and iron pentacarbonyl ($Fe(CO)_5$) in mainstream tobacco smoke. Apart from iron, there is strong epidemiological evidence to implicate the nickel, arsenic, mercury, cadmium, and chromium (and compounds of these elements) present in cigarette smoke in human carcinogenesis. However, no evidence was found to support the theory that $Ni(CO)_4$ or $Fe(CO)_5$ are present in mainstream smoke. Moreover, attempts to promote the formation of metal carbonyls during the combustion of tobacco revealed that such compounds, if formed, do not translocate through the filter. Therefore, loss of metal on combustion of tobacco does not occur via formation of volatile metal carbonyls. The limit of detection for this method was established as 0.1 rl/l for $Ni(CO)_4$ and 0.05 rl/l for $Fe(CO)_5$. (Auth. Abs. Mod.)

84–0672. Brunnemann, K. D.; Hoffmann, D. **IV.5.b. GC-TEA of NDELA From Tobacco Products.** In: Egan, H.; Preussman, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 399-402.

A procedure for the determination of *N*-nitrosodiethanolamine (NDELA) in tobacco smoke is provided. NDELA is separated as ditrimethylsilyl-NDELA at 140°C on a glass column packed with 3 percent OV-225. The retention time for the silylated NDELA is 2 min and the detection limit is 50 ng/injection or 0.5 ng in the mainstream smoke of one cigarette. Quantitation of NDELA is by the isotope dilution method with 2-^{14}C-NDELA. Reagent preparation and method of calculation are also given. (Auth. Abs. Mod.)

84-0673. Brunnemann, K. D.; Hoffmann, D. **II.2.c. *N*-Nitrosodiethanolamine in Tobacco and Mainstream and Sidestream Smoke.** In: Egan, H.; Preussman, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 85-92.

A procedure for the determination of *N*-nitrosodiethanolamine (NDELA) in tobacco products is documented. NDELA is formed from maleic hydrazide-diethanolamine, a widely used sucker growth inhibitor. Tobacco is extracted with ethyl acetate; mainstream or sidestream smoke is collected in a trap containing ethyl acetate and ascorbic acid, which prevents the artefactual formation of NDELA. The extraction product is then chromatographed on silica gel. The NDELA-containing fractions are concentrated, silylated, and analyzed with a modified gas chromatography-thermal electron analysis system. The recovery rate averages about 65 percent, with an observed range of 40 to 80 percent. 2-^{14}C-NDELA is used as a marker. Data concerning reagents and apparatus are included. (Auth. Abs. Mod.)

84-0674. Edwards, W. B., III; McCuen, R. **Preparation of Optically Pure (*R*)-(+)-Nicotine. Studies on the Microbial Degradation of Nicotinoids.** *Journal of Organic Chemistry* 48(15): 2484-2487, July 29, 1983.

(*R*)-(+)-Nicotine of high optical purity (average 99.6 percent) was obtained from (*R,S*)-nicotine by stereoselective microbial degradation of the (*S*)-(—)-nicotine with use of the microorganism *Pseudomonas putida.* Liquid culture results indicated that this organism growing on (*S*)-(—)-nicotine can utilize (*R*)-(+)-nicotine, but at a slower rate. Optimal growth was achieved only when the (*S*)-(—)-nicotine concentration was 1 mg/mL. A concentration of 2 mg/mL exerted a toxic effect on the culture. Studies on related nicotinoids showed the microorganism to be primarily specific for (*S*)-(—)-nicotine. Details of the experimental work are included. (Auth. Abs. Mod.)

84-0675. Elinder, C. G.; Kjellstrom, T.; Lind, B.; Linnman, L.; Piscator, M.; Sundstedt, K. **Cadmium Exposure From Smoking Cigarettes: Variations With Time and Country Where Purchased.** *Environmental Research* 32(1): 220-227, October 1983.

Cadmium (Cd) levels were determined in 26 brands of cigarettes purchased in eight different countries throughout the world and in 16 different samples of cigarettes produced in Sweden between 1918 and 1968. In addition, the amount of Cd released from smoking one cigarette to the particulate phase collected from a smoking simulation machine, corresponding to the amount actually inhaled by a smoker, was determined. The Cd concentration in different brands of cigarettes ranged from 0.19 to 3.0μg Cd/g dry weight, with a general tendency toward lower values in cigarettes from developing countries. No systematic change in the Cd concentration of cigarettes with time was found. The amount of Cd inhaled from smoking one cigarette containing about 1.7μg Cd was estimated to be 0.14 to 0.19μg, corresponding to about 10 percent of the total Cd content in the cigarette. (Auth. Abs.)

84-0676. Gulovali, M. C.; Gunduz, G. **Trace Elements in Turkish Tobacco Determined by Instrumental Neutron Activation Analysis.** *Journal of Radioanalytical Chemistry* 78(1): 189-198, 1983.

Analysis of nine brands of Turkish cigarette tobacco and one brand of pipe tobacco showed that concentration values of potassium and bromine in pipe tobacco are one-half those found in cigarette tobaccos. Values for hafnium, chromium, zinc, sodium, and iron, however, are almost twice the corresponding values for cigarette tobaccos. The factors responsible are genetic variations, differences in soil type, stalk position of the leaves, and saucing material added to pipe tobacco, which may contain up to 30 percent of these additives (licorice, sweetening agents, sugars, and other flavoring materials). Neutron activation analysis of Turkish cigarette tobaccos shows that almost all the elements are present in about the same amounts. Comparisons with American and Iranian cigarette tobaccos show that arsenic concentrations are higher in American cigarettes and undetectable in Iranian brands. It is noted that a large portion of trace elements in cigarette tobacco is left in the ash and only a small fraction is transferred into the smoke. (Auth. Abs. Mod.)

84-0677. Hecht, S. S.; Adams, J. D.; Hoffmann, D. **IV.6. HPLC-TEA of Tobacco-Specific Nitrosamines.** In: Egan, H.; Preussman, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 429-436.

The reagents, apparatus, determination procedure, and method of calculation for the measurement of tobacco-specific nitrosamines are provided. Tobacco-specific nitrosamines are separated by normal phase high pressure liquid chromatography: retention times range from 19.2 to 34.5 minutes. Quantification is by isotope dilution, with 2′-^{14}C-nitrosonornicotine (NNN) as the internal standard. The mean N value for four analyses of NNN in the mainstream smoke of a nonfiltered cigarette was 0.21±0.025 µg/cigarette. (Auth. Abs. Mod.)

84–0678. Hecht, S. S.; Adams, J. D.; Hoffmann, D. **II.2.d. Tobacco-Specific Nitrosamines in Tobacco and Tobacco Smoke.** In: Egan, H.; Preussman, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 93-101.

A procedure for the determination of *N*′-nitrosoanatabine (NAT), *N*′-nitrosonornicotine (NNN), and 4-(methylnitrosoamino)-1-(3-pyridyl)-1-butanone (NNK) in tobacco is described. Tobacco is extracted with aqueous ascorbic acid solution, or mainstream or sidestream smoke is collected in traps containing aqueous ascorbic acid. The use of ascorbic acid prevents artefactual formation of the tobacco-specific nitrosamines. The nitrosamines are then extracted with organic solvents, followed by column chromatography. The appropriate fractions from the columns are then analyzed by combined high pressure liquid chromatography-thermal electron analysis, using 2′-^{14}C-NNN as an internal standard in all analyses. (Auth. Abs. Mod.)

84–0679. Higgins, C. E.; Griest, W. H.; Olerich, G. **Application of Tenax Trapping to Analysis of Gas Phase Organic Compounds in Ultra-Low Tar Cigarette Smoke.** *Association of Official Analytical Chemists Journal* 66(5): 1074-1083, September 1983.

A method well-suited for analyzing gas phase organic compounds in ultra-low-tar delivery cigarette smoke is described. The cigarette is smoked directly through a Cambridge filter and a Tenax trap arranged in series. The components collected on Tenax are analyzed by thermal desorption programmed-temperature gas chromatography. Components are then quantitated using external standards. The method is also applicable to cigarettes with higher tar deliveries and should be useful in analyzing semivolatile smoke components and thermally desorbable particulate matter if applied to whole smoke rather than being restricted to the gas phase. Reliability was demonstrated by determining deliveries of 34 gas-phase components of four ultra-low-tar cigarettes having tar deliveries ranging from <0.01 to 3 mg/cigarette, and for 24 components of five cigarettes delivering 7 to 45 mg/cigarette. (Auth. Abs. Mod.)

84–0680. Hoffmann, D.; Brunnemann, K. D. **IV.2.b. GC-TEA of Volatile Nitrosamines From Tobacco Products.** In: Egan, H.; Preussman, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 363-366.

Procedures for the gas chromatographic-thermal electron analysis (GC-TEA) of tobacco-related volatile *N*-nitrosamines (VNA) are described. Information on the availability of reagents, apparatus needed, and the method for calculation is also provided. The VNA are separated by GC at 200°C on a column packed with Carbowax 20M. Retention times vary between 5 and 21 min. The TEA detection limit is approximately 50 to 200 pg, or 1 to 4 ng in the mainstream smoke of one cigarette. The VNA are quantified via isotope dilution, employing ^{14}C-nitrosodimethylamine. The standard deviation for five analyses was 11 percent in the mainstream smoke of a nonfiltered cigarette.

84–0681. Hoffmann, D.; Brunnemann, K. D.; Webb, K. S. **II.2 Tobacco and Tobacco Smoke (Volatile and Tobacco-Specific Nitrosamines). II.2.b Volatile Nitrosamines in Tobacco and Mainstream and Sidestream Smoke and Indoor Environments.** In: Egan, H.; Preussmann, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 69-83.

The procedures appropriate for analyzing the nitrosamine content of cigarette smoke are detailed, and the necessary apparatus, reagents, precautions, and sampling techniques are described. Tobacco smoke is collected in liquid traps containing ascorbic acid and the trapping section is extracted with dichloromethane. The extract is cleaned by column chromatography and the nitrosamines then determined using gas chromatography-thermal energy analysis. Appropriate modifications are given for nitrosamines in tobacco and polluted indoor environments. (Auth. Abs. Mod.)

84–0682. Hoffmann, D.; Hecht, S. S. **II.2 Tobacco and Tobacco Smoke (Volatile and Tobacco-Specific Nitrosamines). II.2.a General Aspects.** In: Egan, H.; Preussmann, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publications No. 45, 1983, pp. 63-67.

Tobacco smoke contains three types of *N*-nitrosamines: tobacco-specific, volatile, and those deriving from residues of agricultural chemicals. Formation is greatly influenced by the air flow around the burning cone, and all three types are released into the respiratory environment as part of the sidestream smoke. The tobacco-specific nitrosamines are formed by *N*-nitrosation of tobacco alkaloids and include *N*′-nitrosonornicotine (NNN), 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), *N*-nitrosoanatabine (NAT), and trace amounts of *N*-nitrosoanabasine (NAB). NNN, NNK, and NAB are all carcinogens; NAT has not been bioassayed.

Volatile nitrosamines that have been identified in tobacco smoke include *N*-nitrosodimethylamine, *N*-nitrosoethylmethylamine, *N*-nitrosodiethylamine, *N*-nitrosopyrrolidine, and *N*-nitrosomorpholine. The only nitrosamine found thus far that originates in an agricultural chemical is *N*-nitrosodiethanolamine, derived from maleic hydrazine.

84–0683. Kaplan, N. M.; Lee, N. E.; Benowitz, N. L.; Hall, S.; Herning, R. I.; Jacob, P.; Jones, R. T. **Low-Yield Cigarettes. (Letters).** *New England Journal of Medicine* 310(4): 260, January 26, 1984.

The validity of the Federal Trade Commission's (FTC) estimates of nicotine yield/cigarette as discussed earlier (*New England Journal of Medicine* 309(3): 139-142, July 21, 1983) is questioned, noting that the FTC nicotine yield/cigarette could reflect the smoking of a larger number of cigarettes/day by those who smoke low-nicotine cigarettes. Another writer questions the assumption of linear models, suggesting that the data could be fitted to other models, which could yield marked modifications to the conclusions drawn by the original authors, particularly models based on data with so much scatter. The original authors reply that more recent data indicate that nicotine intake averages 50 to 60 percent lower in people switching to very low-yield cigarettes (FTC yields of <0.3 mg). This is interpreted as showing that very-low-yield brands restrict nicotine intake while most of the low-yield cigarettes do not, so that smokers can obtain the dose of nicotine desired. Thus, even with the limitations of the model, it remains clear that there is a marked discrepancy between the 15- to 20-fold difference in nicotine yields predicted by the FTC smoking machine assays comparing the lowest and highest yield brands, and the twofold difference in actual intake of nicotine by smokers. See also 84-0019.

84–0684. Nishikawaji, S.; Fujimori, T.; Matsushima, S.; Kato, K. **Sesquiterpenoids From Flue-Cured Tobacco Leaves.** *Phytochemistry* 22(8): 1819-1820, 1983.

Eight sesquiterpenoids were identified from an essential oil of flue-cured tobacco leaves. One is a novel compound, a tetracyclic sesquiterpene having an α,β-unsaturated ketone and a double bond between C-2 and C-3, and named 2,3-dehydrosolanascone. Also identified were γ-cadinene, δ-cadinene, 2-keto-α-cyperone, solanascone, solavetivone, occidol, and rishitin.

84–0685. Podlejski, J.; Olejniczak, W. **Methods and Techniques in the Research of Tobacco Flavour.** *Nahrung* 27(5): 429-436, 1983.

Research methods for identifying tobacco flavor components, determining aromatic factors, and establishing correlations with organoleptic data are discussed. Basic techniques involve chemical analysis of raw tobacco material, tobacco smoke, and headspace vapors, as well as subjective evaluations of tobacco quality. Laboratory research at a Polish university indicates that both instrumental (chemical) and sensory (taste) tests are necessary to develop the best product. At this facility, tobacco flavor is first evaluated quantitatively to determine total nitrogen, protein, nicotine, water soluble carbohydrates, pectin, cellulose, polypentose, essential oils, resins, polyphenols, organic acids, and water extract pH. Taste tests follow the analyses, and then any necessary modifications to the chemical composition of the cigarette blends are made.

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84–0686. Adaikan, P. G.; Karim, S.M.M.; Kottegoda, S. R.; Ratnam, S. S. **Cholinoreceptors in the Corpus Cavernosum Muscle of the Human Penis.** *Journal of Autonomic Pharmacology* 3(2): 107-111, June 1983.

Nicotine produced contraction in 70 percent and relaxation in 30 percent of corpus cavernosum muscle strips from human penis. The lowest effective concentration was between 0.06 μM and 4.9 μM. Responses were abolished by hexamethonium and tetrodotoxin, while acetylcholine produced contraction (25.7 percent of strips), relaxation (62.9 percent), or biphasic effects (11.4 percent) on corpus cavernosum muscle strips. These effects were antagonized by atropine but were modified by hexamethonium or tetrodotoxin, suggesting the presence of excitatory and inhibitory muscarinic receptors in the human penis. Contractions produced by nicotine were abolished by atropine, whereas the relaxation produced was only partially blocked by atropine. Hence, the receptors mediating relaxation are unlikely to be entirely muscarinic. (Auth. Abs. Mod.)

84–0687. Andersson, K.; Siegel, R. A.; Fuxe, K.; Eneroth, P. **Intravenous Injections of Nicotine Induce Very Rapid and Discrete Reductions of Hypothalamic Catecholamine Levels Associated With Increases of ACTH, Vasopressin and Prolactin Secretion.** *Acta Physiologica Scandinavica* 118(1): 35-40, May 1983.

Results indicate that nicotine given i.v. can, within minutes, induce increases of ACTH, vasopressin, and prolactin secretion in the male rat, giving further evidence for the existence of nicotine-like cholinergic receptors involved in the regulation of these hormones. These changes were associated with rapid reductions of noradrenaline (NA) levels in the subependymal layer of the median eminence, in the nuclei dorsomedialis hypothalami, and in the anterior and posterior periventricular hypothalamic area, as revealed by quantitative microfluorimetrical measurements of catecholamine fluorescence. Intraindividual correlations indicate the involvement of an inhibitory noradrenergic mechanism in the subependymal layer of the median eminence in the regulation of ACTH secretion, the involvement of noradrenergic mechanisms in the posterior periventricular area in the regulation of prolactin secretion and the involvement of dopaminergic mechanisms in the medial palisade zone of the median eminence in the regulation of prolactin secretion. A rapid rise of prolactin secretion seems to be associated mainly with a reduction of NA levels in the posterior periventricular area indicating the existence of a possible facilitatory noradrenergic mechanism in this region regulating prolactin secretion. (Auth. Abs.)

84–0688. Baldwa, V. S.; Gupta, M. C.; Maheshwari, V. D.; Bhansali, A. **Effect of Prolonged Smoking and Alcohol on Lipid Profile, Separately and in Combination.** *Journal of Association of Physicians of India* 31(9): 573-575, September 1983.

Smoking produced an increase in serum cholesterol, triglyceride, and low density lipoprotein cholesterol (LDL-C), and a decrease in high density lipoprotein cholesterol (HDL-C), in apparently healthy subjects who had smoked ≥ 10 cigarettes or ≥ 25 bidis/day for > 10 years. Participants were divided into four age-matched groups: (1) nonalcoholic smokers (n=25); (2) alcoholic smokers (25): (3) alcoholic nonsmokers (25); and (4) nonalcoholic nonsmokers (25 controls). Alcoholics had consumed > 6 oz/week for > 5 years. Alcohol alone resulted in a rise in triglyceride and HDL-C, and a reduction in LDL-C. However, when the two habits were combined, changes were found to be similar to those found for smokers, indicating that any beneficial effect of alcohol consumption is neutralized in smokers. (Auth. Abs. Mod.)

84–0689. Benowitz, N. L. **The Use of Biologic Fluid Samples in Assessing Tobacco Smoke Consumption.** In: Grabowski, J.; Bell, C. S. (Editors). *Measurement in the Analysis and Treatment of Smoking Behavior.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph 48, DHHS Publication No. (ADM) 83-1285, 1983, pp. 6-26.

Review of the source, absorption, metabolism, and disposition kinetics of carbon monoxide (expired and carboxyhemoglobin levels), thiocyanates (serum vs. saliva), nicotine (blood vs. urine), and cotinine (blood vs. urine concentrations) indicates that selection of a biochemical test must be linked to the hypothesis being tested. Kinetic considerations are applied to discuss specificity and sensitivity of the compounds as markers of smoking status, usefulness as quantitative indicators of tobacco smoke consumption, and optimal time for sample collection. If only smoking vs. nonsmoking is assessed, carbon monoxide and/or thiocyanate are inexpensive measurements that provide adequate information. If self-administration of nicotine and/or toxic effects potentially related to nicotine exposure are studied, measurements of nicotine exposure and consumption are required. Measurement of blood concentration of nicotine is necessary to document nicotine exposure; blood concentration of cotinine may be a better measure of daily nicotine consumption. To study potentially carcinogenic effects of tobacco smoke, specific measurements of carcinogen consumption must be developed and validated. (Auth. Abs. Mod.)

84–0690. Bradley, P. B.; Lucy, A. P. **Cholinoceptive Properties of Respiratory Neurones in the Rat Medulla.** *Neuropharmacology* 22(7): 853-858, July 1983.

The effects of iontophoretically applied acetylcholine, the acetylcholine agonists nicotine and muscarine, and the antagonists atropine, dihydro-β-erythroidine (DHβE) and mecamylamine, together with the excitatory amino acids, glutamate and D,L-homocysteic acid (DLH) were examined on the activity of respiratory-related neurons in the rat medulla, and compared with effects on nonrespiratory brain stem neurons. Most neurons were excited by acetylcholine and no inhibitory responses were seen. Glutamate and DLH also acted as excitants but there was a trend for the phasic activity of respiratory neurons to be converted to a tonic discharge. Nicotine excited 74 percent of the neurons to which it was applied, and responses were blocked by DHβE but not by atropine. Muscarine also caused excitation, and responses were blocked by atropine but not by DHβE. Both antagonists blocked acetylcholine-induced excitation but had no effect on responses to glutamate or DLH. Mecamylamine was without effect. It is concluded that the proportion of cholinoceptive respiratory neurons in the rat brain stem is similar to that of nonrespiratory neurons. It seems likely that both nicotinic and muscarinic receptors are present on the majority of respiratory neurons and that both contribute to the response produced by iontophoretically applied acetylcholine. (Auth. Abs.)

84–0691. Cam, G. R.; Bassett, J. R. **The Plasma Levels of ACTH Following Exposure to Stress or Nicotine.** *International Archives of Pharmacology* 264(1): 154-167, July 1983.

Data indicate that, in rats, there is (1) only a single rapid elevation in plasma ACTH levels after stress, nicotine, or ACTH injection, and (2) the concentration of ACTH during its plateau seems to have a more profound effect on the duration, rather than on the magnitude of the second steroid elevation. In the nicotine-induced ACTH secretion, the significantly depressed plateau phase was correlated with the shorter duration of the second plasma steroid rise. Plasma ACTH and corticosterone profiles were measured following intraperitoneal injection of nicotine or ACTH, or following exposure to unpredictable stress. The elevation in plasma corticosterone was biphasic in nature in all cases, whereas plasma ACTH levels demonstrated only a single peak which rapidly declined to a sustained plateau level. The differences between the ACTH and corticosterone profiles, resulting from the three different stimuli, are discussed. (Auth. Abs. Mod.)

84–0692. Cam, G. R.; Bassett, J. R. **The Effect of Acute Nicotine Administration on Plasma Levels of the Thyroid Hormones and Corticosterone in the Rat.** *Pharmacology Biochemistry and Behavior* 19(3): 559-561, September 1983.

While acute administration of nicotine may influence activity and metabolism through its effects on the circulating levels of catecholamines, glucocorticoids, and insulin, it does not appear to alter levels of the thyroid hormones in the short term. The effects of a single intraperitoneal injection of nicotine hydrogen tartrate (200 µg/kg) on the plasma levels of thyroxine, triiodothyronine, and corticosterone were monitored over a 24-hour period. Nicotine did not alter the plasma levels of either of the thyroid hormones, but did produce a significant increase in plasma corticosterone, an effect which peaked at 20 minutes postinjection and lasted for 45 minutes. (Auth. Abs. Mod.)

84–0693. Castonguay, A.; Stoner, G. D.; Schut, H.A.J.; Hecht, S. S. **Metabolism of Tobacco-Specific *N*-Nitrosamines by Cultured Human Tissues.** *Proceedings of the National Academy of Sciences* 80(21): 6694-6697, November 1983.

Data demonstrate that explants of tissues from tobacco smokers' buccal mucosa, trachea, esophagus, bronchi, and peripheral lung, as well as the urinary bladder, have the capacity to metabolize *N'*-nitrosonornicotine (NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) by α-carbon hydroxylation. This metabolic pathway yields alkyldiazohydroxides, which are reactive and DNA-damaging electrophiles. The extent of α-carbon hydroxylation of NNN and NNK in human tissues was only 1/10th to 1/100th of that in animal tissues. Although the levels of α-carbon hydroxylation of NNN among different tissues of the same individual were similar, a 10-fold variation among individuals was observed. Reduction of the NNK carbonyl group was a major metabolic pathway observed with all human explants and may occur in the surface epithelia of the respiratory tract of smokers. These results provide further evidence that tobacco-specific *N*-nitrosamines could play a role in cancers related to the smoking and chewing of tobacco. (Auth. Abs. Mod.)

84–0694. Connelly, M. S.; Littleton, J. M. **Lack of Stereoselectivity in Ability of Nicotine to Release Dopamine From Rat Synaptosomal Preparations.** *Journal of Neurochemistry* 41(5): 1297-1302, November 1983.

Both the naturally occurring (-)-isomer and the synthetic (+)-isomer of nicotine caused release of ^3H from a crude synaptosomal fraction of rat brain preincubated with (^3H)dopamine. The isomers were equipotent in producing this response, which was concentration-dependent, a significant effect on the fractional release of dopamine being observed at 10^{-4} M nicotine. The effect did not appear to be the result of synaptosomal damage, as levels of the intrasynaptosomal marker lactate dehydrogenase did not increase in the supernatant. Nicotine-induced release was inhibited by removal of external Ca^{2+} and by the presence in vitro of pempidine (230 µM). Neither hexamethonium (500 µM) in vitro nor the chronic administration of (-)-nicotine in vivo had any effect on the nicotine-induced release of (^3H)dopamine. It is concluded that nicotine exerts this effect via a presynaptic nicotinic receptor of the ganglionic type, but that this receptor differs from that in the periphery by showing a relative lack of stereospecificity. There is no evidence of a functional "down regulation" in this receptor on chronic exposure to nicotine in vivo. (Auth. Abs.)

84–0695. Emley, G. S.; Hutchinson, R. R. **Unique Influences of Ten Drugs Upon Post-Shock Biting Attack and Pre-Shock Manual Responding.** *Pharmacology Biochemistry and Behavior* 19(1): 5-12, July 1983.

Subcutaneous injections of nicotine in four squirrel monkeys elevated lever press responding while depressing bite responding across a portion of the dosage range. The two temporally and topographically different responses in a single organism were elicited by delivery of a fixed-time, response-independent electric tail shock that generated bites on a rubber hose immediately following shock and manual responses on a lever immediately preceding shock. d-Amphetamine, cocaine, and caffeine each elevated both bite and lever press responses. Nicotine, chlorpromazine, chlordiazepoxide, and diazepam each elevated lever press responding while depressing bite responding; phenobarbital, alcohol, and morphine depressed both bite and lever press responses but lever pressing was selectively more depressed than biting. Results parallel previous research with these drugs on other measures of aggression and on other behavioral paradigms. The responses are contingency-free so that the effect of a drug does not interact with response-produced environmental consequences. The recording of two separate responses related to distinct emotional states allows for the objective measurement of selective and differential drug effects. (Auth. Abs. Mod.)

84–0696. Ercan, Z. S.; Zengil, H.; Akar, F.; Turker, R. K. **Possible Prostacyclin-Mediated Vascular Effect of Angiotensin II in the Isolated Perfused Rat Lung.** *Prostaglandins Leukotrienes and Medicine* 12(1): 77-83, September 1983.

Addition of nicotine (N) to the perfusion medium caused a significant increase in perfusion pressure (PP) to angiotensin II (A II) without altering that to angiotensin I (A I). Injecting A I and A II through the pulmonary artery increased PP of the isolated perfused rat lung and caused a contraction when the venous outflow was superfused over rat ascending colon (RC). Addition of ZK 36374, a stable analog of prostacyclin (PGI_2), to the medium prevented the potentiating effect of N on the A II pressor response. Neither N nor ZK 36374 altered the superfused RC responses to A I- and A II-injected venous effluent. Lysine acetylsalicylate (ASA), however, caused a potentiation in the pressor response to A I. The response of venous effluent superfused RC to A I was also found to be potentiated by ASA. ASA failed to alter the responses to A II. It is concluded that A II is a potent activator for the biosynthesis of PGI_2 in the pulmonary vascular bed. Moreover, PGI_2 does not affect angiotensin-converting enzyme activity in the lung circulation while other stable metabolites of arachidonic acid can inhibit the conversion of A I to A II. (Auth. Abs. Mod.)

84–0697. Everson, R. B.; Flack, P. M.; Sandler, R. S. **Urinary Excretion of Mutagens in Cirrhosis: Limited Evidence of an Association.** *Environmental Research* 32(1): 118-126, October 1983.

Although bacterial mutagenesis assays have detected mutagenic substances in urine from smokers and patients treated with certain medications, these assays have not found mutagens in body fluids of subjects without such exposures. A recent report, however, suggested that mutagenic substances were present in the urine of unexposed individuals with cirrhosis. Since this observation could have important implications for the metabolism of potentially toxic substances and might help explain the high incidence of hepatoma among patients with cirrhosis, the reproduction of those findings was attempted. Three groups of patients were studied: (1) nine patients with cirrhosis of the liver; (2) nine subjects with other liver diseases; and (3) volunteer and patient smokers with no clinical history or other evidence of liver disease. In contrast with the earlier report, no evidence of mutagenic substances in urine samples from unexposed patients with cirrhosis was observed. While differences between the studies might be attributed to the subjects tested, it seems possible that the mutagenic activity reported previously was due to technical interference with the testing procedure by nonmutagenic materials in urine rather than the presence of mutagens. It is concluded that in human studies, the best established instances of the excretion of mutagens clearly detectable by bioassay remain those associated with smoking and drug exposures. (Auth. Abs. Mod.)

84–0698. Fraser, H. S.; Tibbits, R.C.D. **Variation in Response to Drugs: Part II. Environmental and Nutritional Variables.** *West Indian Medical Journal* 32(2): 66-74, June 1983.

In a summary of the major environmental and nutritional sources of variation in drug response, cigarette smoking research is reviewed that deals with the effect of smoking on the biotransformation of several drugs. Pertinent data from studies on the role of smoking in pharmacokinetics, the enzyme-inducing properties of cigarette smoke, the effect of smoking on drug elimination rates and plasma concentrations, and smoking's relationship with analgesia efficacy, are listed. From results of such investigations, it is concluded that smokers usually require higher doses of oxidized drugs than do nonsmokers. Environmental and nutritional influences on drug response reviewed, in addition to smoking, include alcohol consumption, marihuana, oral contraceptives, agricultural and environmental contaminants, malnutrition, anemia, dietary intake, and genetic factors.

84–0699. Gibson, G. E.; Pelmas, C. J.; Peterson, C. **Cholinergic Drugs and 4-Aminopyridine Alter Hypoxic-Induced Behavioral Deficits.** *Pharmacology Biochemistry and Behavior* 18(6): 909-916, 1983.

Nicotine (5.5±0.7), or the muscarinic agonist arecoline (5.4±0.5), improved performance on the tightrope test and, when the drugs were combined, the score was even higher (8.2±1.0). The effects of cholinergic drugs and 4-aminopyridine, an enhancer of acetylcholine (ACh) release, were examined in hypoxic mice to test the hypothesis that decreased ACh metabolism during hypoxia is behaviorally important. After mice were rendered hypoxic by 150 mg/kg $NaNO_2$, tightrope test scores were reduced from 13.2±0.2 to 2.8±0.3. 4-Aminopyridine partially reversed the scores of hypoxic mice (7.3±0.7). Physostigmine improved performance (7.9±0.4) when it was given before $NaNO_2$, an effect blocked by pretreatment with atropine (3.5±0.4) or mecamylamine (2.4±0.6). Neostigmine (0.002-0.2 mg/kg) was ineffective. Performance also improved if atropine (7.0±0.5) or mecamylamine (7.2±0.5) was given before $NaNO_2$. Neither epinephrine (0.002-2.0 mg/kg) nor norepinephrine (0.00002-2.0 mg/kg) improved performance by hypoxic mice. Results suggest that hypoxia produces a behaviorally important impairment of the cholinergic system, perhaps through a primary alteration of acetylcholine release. (Auth. Abs. Mod.)

84–0700. Grau, T.; Romero, A.; Sacristan, A.; Ortiz, J. A. **Study on the Protection of CDP-Choline Against Nicotine Intoxication.** *Arzneimittel Forschung* 33(II, 7a): 1025-1026, 1983.

The toxic cholinergic effects of nicotine in mice were attenuated by pretreatment with cytidine diphosphate choline (CDP-choline). CDP-choline, 1 g/kg in 0.25 percent agar, was orally administered to mice for 4 days; controls received agar alone. After pretreatment, increasing doses of nicotine bitartrate were given i.v. At the 1.32 mg/kg nicotine dose, mortality among the pretreated group was 12.5 percent, compared with 82.35 percent among controls. The LD_{50} values were 1.67 and 1.03 mg/kg, respectively, a statistically significant difference.

84–0701. Grunberg, N. E.; Morse, D. E.; Barrett, J. E. **Effects of Urinary pH on the Behavioral Responses of Squirrel Monkeys to Nicotine.** *Pharmacology Biochemistry and Behavior* 19(3): 553-557, September 1983.

The effects of nicotine were examined on the responding of two squirrel monkeys under a multiple fixed-interval (FI), fixed-ratio (FR) schedule of stimulus-shock termination when nicotine was administered alone or together with the gastric administration of an acidifer (ammonium chloride) or an alkalinizer (sodium bicarbonate). Responding under the FI schedule was increased markedly across a range of doses of nicotine (0.02 to 0.20 mg/kg). Responding under the FR was increased to a lesser extent by the lower doses of nicotine (0.02 to 0.05 mg/kg), and was decreased by doses of nicotine that increased responding under the FI (0.10 to 0.20 mg/kg). Generally, administration of the acidifier attenuated the effects of nicotine while administration of the alkalinizer potentiated those effects. These findings support the argument that changes in cigarette smoking under conditions that alter urinary pH involve nicotine per se. In addition, a new interpretation of the relationship between urinary pH and cigarette smoking is offered. (Auth. Abs. Mod.)

84–0702. Herning, R. I.; Jones, R. T.; Bachman, J. **EEG Changes During Tobacco Withdrawal.** *Psychophysiology* 20(5): 507-512, September 1983.

Tobacco smoking decreases theta and alpha power and increases the dominant alpha frequency in smokers deprived of cigarettes for 10 to 17 hours. The slow alpha frequency in deprived smokers has been attributed to either characterological differences between smokers and nonsmokers or nicotine withdrawal. Studies finding slower alpha in deprived smokers were unable to replicate previous findings of decreased alpha abundance after smoking, and did not consider the importance of increased theta as a withdrawal sign. Theta and alpha power increased with tobacco deprivation but were decreased for 30 to 90 min by nicotine cigarettes in 11 smokers studied for 1 day and 7 smokers studied for 2 days while smoking placebo and nicotine cigarettes. The dominant alpha frequency in deprived (compared with nondeprived) smokers was slower, but decreases in frequency were not always statistically significant. (Auth. Abs.)

84–0703. Ishikawa, T.; Nagashima, C. **Jinjin Hishitsuchu no Kadomiumu o Taisho to Shita Seibutsu Monitaringu. [A Biological Monitoring on Cadmium in Normal Human Kidney Cortex.]** *Annual Report of Tokyo Metropolitan Research Laboratory of Public Health* 33: 328-330, 1982, Japanese.

Analysis of autopsied human kidney cortex tissues at Tokyo Metropolitan Hospital showed no significant impact on cadmium (Cd) levels attributable to smoking. Cd concentrations for subjects in their twenties averaged 27.5 mg/kg, while subjects in their thirties averaged 67.2 mg/kg. However, no significant difference in Cd levels was found in subjects aged 30 to over 70. Since one Japanese cigarette contains approximately 1 microgram Cd, smoking was expected to increase kidney levels significantly, but no significant relationship was found. Japan has more than twice the environmental Cd concentration of the eight countries (Belgium, China, India, Israel, Japan, Sweden, the United States, and Yugoslavia) for which data were available. It is suggested that some source of Cd pollution previously present in Japan has been eliminated or reduced, resulting in lower tissue Cd levels in those under age 30.

84–0704. Ito, Y.; Mizuno, S.; Komori, Y.; Otani, M.; Fukaya, M.; Shima, S.; Sarai, S. **Bojigyosho Jugyo-in no Ketsuseishishitsu-chi ni Oyobosu Kitsuen, Inshu Oyobi Setsushu Eiyoso no Eikyo. [Effects of Smoking, Alcohol Drinking and Nutrient Intake on Serum Lipid Values of Workers in an Industry.]** *Journal of Science and Labour* 59(9): 401-417, September 1983, Japanese.

Cigarette smoking elevated total cholesterol values and decreased high density lipoprotein cholesterol (HDL-C) in industrial workers studied for the relationship between serum lipid values and daily living factors such as alcohol consumption, smoking, and diet. Total cholesterol was correlated with protein and fat intake and inversely correlated with consumption of cereals, legumes, and miso. HDL-C was significantly correlated with total caloric intake, protein, fat, and fish consumption, but negatively correlated with intake of carbohydrates, cereals, and sugar. Alcohol consumption was significantly correlated with HDL-C values, but not with total cholesterol values. Thus, higher consumption of vegetables, daily exercise, and moderate alcohol consumption apparently contribute to an improvement in levels of total cholesterol and HDL-C in the industrial workers.

84–0705. Janzon, L.; Berntorp, K.; Hanson, M.; Lindell, S.-E.; Trell, E. **Glucose Tolerance and Smoking: A Population Study of Oral and Intravenous Glucose Tolerance Tests in Middle-Aged Men.** *Diabetologia* 25(2): 86-88, August 1983.

A comparison of normal-weight male smokers, ex-smokers, and nonsmokers born between 1926 and 1929 indicated that smoking has a clinically insignificant influence on both oral and intravenous glucose tolerance tests. No difference was found when comparing fasting glucose in nonsmokers, ex-smokers, and smokers. In the oral glucose tolerance test, plasma glucose at 40 and 60 min increased stepwise from 8.7 and 7.4 mmol/l in 325 nonsmokers to 8.9 and 7.5 mmol/l in 300 ex-smokers, 9.2 and 7.9 mmol/l in 681 smokers, and 9.7 and 8.2 mmol/l in 148 heavy smokers. Blood glucose levels at 120 min were inversely arranged. Plasma insulin at 120 min was lower in heavy smokers (16.2 mU/l) than in nonsmokers (24.8 mU/l). However, the mean intravenous glucose tolerance test k-value was lower in smokers than in nonsmokers, and k-values below 1.0 were twice as common in smokers (30 percent) as in nonsmokers. (Auth. Abs. Mod.)

84–0706. Jarvik, M. E.; Gritz, E. R.; Rose, J. E. **Cigarette Smoking Behavior in Conjoined Twins.** *Acta Geneticae Medicae et Gemellologiae* 32(3/4): 239-243, 1983.

Cigarette smoking in a pair of 32-year-old female craniopagus twins was studied to determine how a transfer of products of smoking occurred between the twins. The twins began smoking when they were 21 years of age, and smoked about a pack of cigarettes/day. Alternately and independently, one twin smoked a nicotine-free cigarette, then the second twin smoked a cigarette containing nicotine. The procedure enabled the investigators to study the migration of nicotine and carbon monoxide from one twin to the other. Salivary determination provided a noninvasive method of measuring cross-circulation in conjoined twins. Measurements of salivary nicotine indicated that, although nicotine levels rose following smoking, there was relatively little transfer from one twin to the other through the circulation. (Auth. Abs. Mod.)

84–0707. Jarvis, M. J.; Russell, M.A.H.; Feyerabend, C. **Absorption of Nicotine and Carbon Monoxide From Passive Smoking Under Natural Conditions of Exposure.** *Thorax* 38(11): 829-833, November 1983.

Nicotine and cotinine levels in plasma, saliva, and urine, and carbon monoxide in expired air were increased in seven nonsmokers exposed to tobacco smoke under natural conditions for 2 hours in a public house (pub). The average concentrations of carbon monoxide, nicotine, and cotinine in nonsmokers, expressed as percentages of the levels found in smokers, were 15.7, 7.5, and 1.8 percent, respectively. Although the increase of carbon monoxide in expired air of 5.9 ppm was similar to increases in smokers after a single cigarette, the amount of nicotine absorbed was between one-tenth and one-third of the amount taken in from one cigarette. Since this represented a relatively extreme acute natural exposure, any health risks of passive smoking probably depend less on quantitative factors than on qualitative differences between sidestream and mainstream smoke. (Auth. Abs. Mod.)

84–0708. Kaelin, A. C.; Cummings, A. J. **A Study of Monooxygenase Activity in Human Placental Homogenates: *In Vitro* Behaviour Towards a Number of Substrates and Inhibitors.** *Biochemical Pharmacology* 32(16): 2421-2426, August 15, 1983.

The in vitro effects of cimetidine, metyrapone, SKF-525A, and α-naphthoflavone on the monooxygenase activity in human placental tissue were determined by indirect fluorimetric assay methods in placental homogenates from five maternal smokers. The cytochrome P-448-associated monooxygenase enzyme system is induced in placentas as a result of maternal cigarette smoking during pregnancy. The inhibitor concentrations producing half-maximum inhibition (I_{50} values) were calculated for the *O*-deethylation of 7-ethoxycoumarin and 7-ethoxyphenoxazone, and the hydroxylation of 2,5-diphenyloxazole. Results indicate that cimetidine is a weak inhibitor of the placental monooxygenase system, resembling metyrapone and SKF-525A in its effects rather than α-naphthoflavone. Characterization of the behavior of the three substrates toward placental monooxygenase activity indicates a much greater enzymic affinity for 7-ethoxyphenoxazone than for 7-ethoxycoumarin or 2,5-diphenyloxazole. (Auth. Abs. Mod.)

84–0709. Kivela-Ikonen, P.; Hanninen, O.; Kalliokoski, P.; Koivusaari, U. **Influence of Fly Ash and Charcoal on the Inducer Activity of Benzo(*a*)pyrene and Smoke Condensate in the Gut Mucosa of the Rat.** *Environmental Research* 32(1): 1-7, October 1983.

Administration of cigarette smoke condensate to rat intestine caused a slight but insignificant increase in mucosal benzo(*a*)pyrene (BaP) hydroxylase activity. Activated charcoal eliminated the smoke condensate effects on enzyme activity. Conversely, BaP alone increased activity severalfold within 24 hours; it returned to control levels after 2 to 3 days. Fly ash did not influence BaP-induced activity, but activated charcoal blocked the biological activity of BaP up to 50 mg/g in all gut segments studied. Cigarette smoke condensate required a concentration 150 times higher to obtain a result similar to BaP (BaP ratio in condensate was about 24). Mucosal 7-ethoxyresorufin deethylase responded to BaP and condensate in the same manner as BaP hydroxylase.

84–0710. Lewis, D.; Capell, H. A.; McNeil, C. J.; Iqbal, M. S.; Brown, D. H.; Smith, W. E. **Gold Levels Produced by Treatment With Auranofin and Sodium Aurothiomalate.** *Annals of the Rheumatic Diseases* 42(5): 566-570, October 1983.

In 63 rheumatoid arthritis patients, treated with either sodium aurothiomalate (Myocrisin), auranofin, or placebo, the hemolysate gold level achieved was dependent on the number of cigarettes smoked, and the dose—response relationship was statistically significant ($p < 0.001$). Gold levels in whole blood, plasma, and hemolysate were measured serially along with clinical and laboratory parameters of efficacy. Auranofin produced a higher ratio of hemolysate to plasma gold than Myocrisin, and it appeared that the affinity of the red cell for gold was reduced during therapy with auranofin. Gold levels did not correlate with changes in the pain score, erythrocyte sedimentation rate, and C-reactive protein, nor with the development of toxicity. In the auranofin group, the correlation with amount smoked was not found, but the hemolysate gold level was higher for smokers than for nonsmokers. With auranofin, there appears to be a natural mechanism for cellular uptake that is missing with Myocrisin. In smokers, either the cell membrane is disturbed, or, more likely, the extra thiocyanate or cyanide ligands added to the plasma react with the gold to form a compound that is readily transported across the membrane. Since plasma and lysate gold levels do not correlate with therapeutic response and there appears to be no evidence that smoking enhances the action of Myocrisin, measurement of intracellular or extracellular gold levels does not seem to be useful in monitoring gold therapy per se. (Auth. Abs. Mod.)

84–0711. Linnainmaa, K. **Sister Chromatid Exchanges Among Workers Occupationally Exposed to Phenoxy Acid Herbicides 2,4-D and MCPA.** *Teratogenesis, Carcinogenesis, and Mutagenesis* 3(3): 269-279, 1983.

A slight but statistically significant difference in sister-chromatid exchange (SCE) was seen between smokers ($n = 16$) and nonsmokers ($n = 19$) in blood cultures from samples taken from male herbicide workers before, during, and after a spraying period. Smokers had mean values averaging 1 to 2 SCEs/cell higher than nonsmokers. SCE induction was studied in the peripheral lymphocytes of workers spraying foliage with phenoxy acid herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2-methyl-4-chlorophenoxyacetic acid (MCPA) or their mixtures. Concentrations of 2,4-D and MCPA, measured in sprayers' urine samples after exposure, ranged from 0.00 to 10.99 mg/l (mean 1.80, standard deviation (SD) 2.85), and from 0.00 to 10.93 mg/l (mean 2.80, SD 3.73) for nonsmokers and smokers, respectively. Several subjects in both groups had no detectable 2,4-D or MCPA urine concentrations, indicating proper use of protective equipment. Data showed no observable trend toward higher mean SCE frequencies in relationship to higher exposure levels for smokers or nonsmokers. Results indicate that 2,4-D and MCPA do not act as direct DNA-damaging agents.

84–0712. Maron, M. B.; Crowe, T. D.; Braughler, J. M. **Cigarette Smoke Causes Acute Fluctuations in the Cyclic GMP Content of the Isolated Intact Lung.** *Respiration* 45(1): 39-44, January-February 1984.

The injection of filtered smoke into the airway during inspiration resulted in a strikingly rapid but transient dose—response increase in lung guanosine 3′,5′-monophosphate (cyclic GMP) content. The largest increase was observed 2 sec after injection (the earliest obtainable sample after exposure), which represented the peak of the inspiration in which the smoke was delivered. By 30 sec after smoke exposure, the cyclic GMP content was generally less than half of the observed peak response. Complete recovery occurred by 2 min after injection. Repeated smoke exposure of the same lung generated similar lung responses. The adenosine monophosphate content of lungs was not altered by any concentration of cigarette smoke tested. In addition, the smoke-related changes in lung cyclic GMP content were not associated with any measurable change in lower lung arterial or airway pressures. (Auth. Abs. Mod.)

84–0713. Merrett, J.; Peatfield, R. C.; Rose, F. C.; Merrett, T. G. **Food Related Antibodies in Headache Patients.** *Journal of Neurology, Neurosurgery, and Psychiatry* 46(8): 738-742, August 1983.

A high proportion of smokers may explain the high IgE levels found among cluster headache patients. To test whether food allergy is a major cause of migraine, sera were collected from 208 adults: 74 with dietary migraine, 45 with nondietary migraine, 29 with cluster headache, and 60 controls. No significant differences were identified between any of the groups, with the one exception that cluster headache patients had significantly raised levels of total serum IgE. Of 19 male cluster headache subjects for whom smoking data were available, the 16 who smoked >5 cigarettes/day had a mean IgE titre of 65.9 units. The three who smoked <5 cigarettes/day had a mean IgE level of 16.8 units. Mean IgG4 titres to cheese, milk, and chocolate were very similar in the dietary and nondietary migraine patients and the control subjects, although lower in the cluster headache patients. There was, therefore, no evidence that the food intolerance often associated with migraine headaches is associated with a conventional allergic mechanism. (Auth. Abs. Mod.)

84–0714. Modak, A. T.; Alderete, B. E. **Nicotine Potentiates Sodium Pentobarbital But Not Ethanol Induced Sleep.** *Substance and Alcohol Actions/Misuse* 4(4): 321-325, 1983.

The interaction between nicotine and the central nervous system depressants, sodium pentobarbital and ethyl alcohol, was investigated in Sprague-Dawley mice. Dose—response data were obtained from groups of 8 to 12 mice receiving i.p. injections of 2.5 and 5 mg/kg nicotine or 50 mg/kg sodium pentobarbital 15 minutes prior to the i.p. administration of 3 gm/kg ethanol. Nicotine potentiated, in a dose-dependent manner, the sleep time induced by sodium pentobarbital, but not that induced by ethanol. Mecamylamine, a nicotinic receptor antagonist, blocked the nicotine-induced increase in sleep time. Atropine itself reduced sleep time, but did not change the nicotine effect. It is hypothesized that the central and peripheral nicotinic receptors play an important role in potentiating sodium pentobarbital-induced sleep time. (Auth. Abs. Mod.)

84–0715. Ojewole, J.A.O. **Effects of Drugs and Electrical Stimulation on Rainbow Lizard (Agama agama Linn.) Isolated Gastrointestinal Tract Smooth Muscles.** *Methods and Findings in Experimental and Clinical Pharmacology* 5(5): 299-310, 1983.

Nicotine neither contracted nor relaxed isolated alimentary canal segments from the rainbow lizard in an examination of the effects of various autonomic drugs and transmural electrical stimulation on isolated esophagus, stomach, duodenum, ileum, colon, and rectum. Acetylcholine and its natural or synthetic analogs contracted the esophagus, duodenum, ileum, colon, and rectum (but not the stomach) in a concentration-dependent manner. Atropine exhibited characteristics of competitive antagonism against acetylcholine on lizard isolated rectum, while low to medium concentrations of eserine potentiated acetylcholine-induced but not carbachol-induced contractions. Adrenaline and its analogs dose dependently relaxed isolated segments from various regions, but not from the stomach. The catecholamine-induced relaxations of the lizard gut smooth muscles were inhibited or abolished by low concentrations of phentolamine. Repetitive transmural electrical excitation induced tetrodotoxin- and atropine-resistant, frequency-dependent contractions of all isolated segments except the stomach. Findings that medium to high concentrations of physostigmine, dimethylphenylpiperazinium, and nicotine failed to elicit intrinsic nerve-mediated contractions, and that tetanic transmural electrical stimulation caused tetrodotoxin-resistant contractions of the rainbow lizard isolated gut muscles, strongly suggest that the autonomic innervation of this reptilian alimentary canal differs from that of a conventional mammalian (e.g., guinea pig) gastrointestinal tract. (Auth. Abs. Mod.)

84–0716. Peterson, D. F.; Coote, J. H.; Gilbey, M. P.; Futuro-Neto, H. A. **Differential Pattern of Sympathetic Outflow During Upper Airway Stimulation With Smoke.** *American Journal of Physiology* 245(3): R433-R437, September 1983.

Cigarette smoke stimulation caused decreased sympathetic discharge to the heart and increased sympathetic discharge to the kidney. In control rabbits (n=4), 50 ml of cigarette smoke perfused by cannula across the upper respiratory irritant receptors and out through the nares caused apnea, decreased heartbeat (-116 beats/min), and increased blood pressure (+33 mm mercury). Activity in the renal nerve increased to 248 percent, and activity in the cardiac nerves decreased to 62 percent, of baseline. After paralysis was induced with gallamine triethiodide and the experiment repeated using artificial respiration, nerve activity responses were still pronounced (renal 178 percent and cardiac 66 percent of baseline). In barodenervated rabbits (n=4), neural responses to smoke were similar to those observed in animals with intact baroreceptors, and there was no significant change after paralysis and artificial respiration. It is concluded that cigarette smoke stimulation causes a differential pattern of sympathetic discharge, and that the observed responses are not due to secondary adjustments through arterial baroreceptors, chemoreceptors, or pulmonary stretch receptors. (Auth. Abs. Mod.)

84–0717. Pratt, J. A.; Stolerman, I. P.; Garcha, H. S.; Giardini, V.; Feyerabend, C. **Discriminative Stimulus Properties of Nicotine: Further Evidence for Mediation at a Cholinergic Receptor.** *Psychopharmacology* 81(1): 54-60, August 1983.

Rats were trained to discriminate subcutaneously administered nicotine (0.4 mg/kg) from saline in a standard two-bar operant conditioning procedure with food reinforcement. The response to nicotine was dose-related and at the median effective dose of 0.14 mg/kg, plasma nicotine concentrations were similar to those reported previously for cigarette smokers who inhale. The nicotine analogs anabasine and cytisine increased nicotine-appropriate responding in a dose-related manner. Animals predominantly responded on the saline-associated lever when administered drugs from a range of pharmacological classes, even at doses that were sufficiently large to reduce the overall number of responses. The results confirm that the nicotine discriminative stimulus is highly specific. Previous work has shown anabasine and cytisine to be active at nicotinic-cholinergic binding sites in rat brain. The finding that there is some correlation between the behavioral effects of these compounds and their actions at the nicotine binding site may indicate that the nicotine cue is mediated through a cholinergic receptor. (Auth. Abs.)

84–0718. Priola, D. V.; Sanchez-Wilson, C.; Curtis, M. B. **Pattern of Cholinergic Sensitivity Changes Following Partial Cardiac Denervation.** *Proceedings of the Western Pharmacology Society* 26: 165-167, 1983.

Data showed no discernible pattern in the nicotinic responses of right or left vagotomized dogs compared with controls, and no indication of increased sensitivity of the intrinsic cardiac nerves in any chamber in either group of partially denervated animals. While individual animals frequently showed a strikingly sensitive atrial or ventricular response to nicotine, there was no consistent pattern to these isolated instances of apparent supersensitivity. Definite dose—response relationships for the negative inotropic responses of all four chambers were shown for nicotine and acetylcholine, although the latter was not as clear.

84–0719. Romert, L.; Jenssen, D. **Rabbit Alveolar Macrophage-Mediated Mutagenesis of Polycyclic Aromatic Hydrocarbons in V79 Chinese Hamster Cells.** *Mutation Research* 111: 245-252, 1983.

Rabbit pulmonary alveolar macrophages (PAM) were used as a metabolizing device in combination with V79 Chinese hamster cells as a mutational indicator system. The capacity for metabolic activation of benzo(*a*)pyrene (BaP), its 7,8-diol, and 2-aminoanthracene by PAM was investigated. Because of the high variation between different PAM preparations, a statistically significant effect of the three compounds could only be demonstrated in a series of four or five experiments. When the ability of PAM to metabolize BaP and the 7,8-diol to mutagenic products was compared with that of primary embryonic fibroblasts from Syrian hamsters, PAM were found to be one-tenth as efficient. Experiments were performed to determine how the phagocytic process could affect the metabolic activation of polycyclic aromatic hydrocarbons. The results showed that PAM-mediated mutagenesis of the 7,8-diol was enhanced 5- to 10-fold if PAM were fed with opsonized particles. The mechanism by which the phagocytosis of PAM enhanced the mutagenicity of 7,8-diol, as detected in cocultivated V79 cells, was not determined. (Auth. Abs.)

84–0720. Rowell, P. P.; Hurst, H. E.; Marlowe, C.; Bennett, B. D. **Oral Administration of Nicotine: Its Uptake and Distribution After Chronic Administration to Mice.** *Journal of Pharmacological Methods* 9(4): 249-261, June 1983.

In an investigation of the suitability of administering nicotine to experimental animals by inclusion in the drinking water, it was found that, after an initial accommodation period of several weeks, nicotine could be administered up to a concentration of 100 μg/ml with no decrease in fluid intake or weight gain compared with controls. An analysis of the steady-state plasma levels and distribution of nicotine was made in mice that had received 60 μg/ml nicotine in drinking water. The average daily dose of nicotine received was 17.2 mg/kg, and the steady-state plasma level was 34.4 Mg/ml, representing 6 percent of the total compound present at steady-state as determined by thin-layer chromatography. Whole-body autoradiography and direct tissue counting in mice that had received (methyl-^{14}C)-nicotine orally demonstrated that nicotine accumulates in a number of areas, particularly the salivary gland, nasal epithelium, uterus, and liver. There was relatively little material in the blood or brain. Results indicate that ad libitum oral administration is an acceptable method for maintaining experimental animals on nicotine for long periods of time. (Auth. Abs. Mod.)

84–0721. Selkirk, J. K.; Nikbakht, A.; Stoner, G. D. **Comparative Metabolism and Macromolecular Binding of Benzo(*a*)pyrene in Explant Cultures of Human Bladder, Skin, Bronchus and Esophagus From Eight Individuals.** *Cancer Letters* 18(1): 11-19, February 1983.

Benzo(*a*)pyrene (BaP) metabolism and macromolecular binding were studied in explants from four tissues (bladder, skin, bronchus, and esophagus) from eight donors sampled within 4 hours after death. Explants were incubated with (^{3}H)benzo(*a*)pyrene for 24 hours; the metabolites were then extracted and analyzed by high pressure liquid chromatography. Fibroblasts were grown from explants from two patients and incubated with BaP. Metabolite profiles were qualitatively the same for explants and fibroblasts with similar product ratios, although fibroblasts were less active in BaP metabolism. DNA binding studies showed a broad variance between patients and tissues; the relative distribution was widest in bladder, followed by skin, bronchus, and esophagus. (Auth. Abs.)

84–0722. Seyler, L. E., Jr.; Fertig, J.; Pomerleau, O.; Hunt, D.; Parker, K. **The Effects of Smoking on ACTH and Cortisol Secretion.** *Life Sciences* 34(1): 57-65, 1984.

The relationships among changes in plasma nicotine, adrenocorticotrophic hormone (ACTH), and cortisol secretion after smoking were investigated in 10 male subjects who smoked cigarettes containing 2.87 mg nicotine and 0.48 mg nicotine. No increases in cortisol or ACTH were detected after smoking 0.48 mg nicotine cigarettes. Cortisol rises were significant in 11 of 15 instances after smoking 2.87 mg nicotine cigarettes, but ACTH rose significantly in only 5 of the 11 instances where cortisol increased. Each ACTH rise occurred in a subject who reported nausea and was observed to be pale, sweaty, and tachycardic. Peak plasma nicotine concentrations were not significantly different in sessions when cortisol rose with or without ACTH increases, but cortisol increases were significantly greater in nauseated than in nonnauseated smokers. Data suggest that smoking-induced nausea stimulates cortisol release by stimulating ACTH secretion, and that cortisol secretion in nonnauseated smokers may occur through a non-ACTH mechanism. It is not clear whether nicotine or some other stimulus inherent in smoking is responsible for cortisol secretion without ACTH secretion. (Auth. Abs.)

84–0723. Siegel, R. A.; Andersson, K.; Fuxe, K.; Eneroth, P.; Lindbom, L.-O.; Agnati, L. F. **Rapid and Discrete Changes in Hypothalamic Catecholamine Nerve Terminal Systems Induced by Audiogenic Stress, and Their Modulation by Nicotine-Relationship to Neuroendocrine Function.** *European Journal of Pharmacology* 91(1): 49-56, July 15, 1983.

Stress enhanced the rapid but variable increase in vasopressin secretion induced by nicotine, suggesting a mechanism by which stress combined with smoking can contribute to development of increased arterial blood pressure and sustained hypertension. The effects of acute audiogenic stress, with or without simultaneous nicotine treatment (0.3 mg/kg i.v.), on catecholamine levels in discrete dopamine and noradrenaline (NA) nerve terminal systems of the hypothalamus, and on the secretion of adenohypophyseal hormones and corticosterone, were studied using quantitative microfluorometric evaluations of catecholamine stores and radioimmunoassays for determining serum hormone levels. Audiogenic stress and nicotine induced very rapid and discrete decreases in NA levels in the subependymal layer (SEL), in the parvocellular part of nuclear paraventricular hypothalamic (PA FP), and in the posterior periventricular hypothalamic systems (PV II); the decreases were apparent 2 minutes following the onset of treatment. Increases of arterial blood pressure were observed after nicotine treatment, but could not have been a major factor in producing the changes in catecholamine levels. These changes in NA levels may be related to the nicotine- and stress-induced increases of ACTH (SEL and PA FP) and prolactin secretion (PV II) found. (Auth. Abs.)

84–0724. Stanwell-Smith, R.; Thompson, S. G.; Haines, A. P.; Ward, R. J.; Cashmore, G.; Stedronska, J.; Hendry, W. F. **A Comparative Study of Zinc, Copper, Cadmium, and Lead Levels in Fertile and Infertile Men.** *Fertility and Sterility* 40(5): 670-677, November 1983.

Although smokers had higher mean levels of blood cadmium than nonsmokers (0.22 nmol/l vs. 0.08 nmol/l), there was no evidence that higher cadmium concentrations affect smokers' fertility. Eighty infertile men (45 percent smokers) and 38 men of known fertility (32 percent smokers) were studied to determine the importance of zinc, copper, cadmium, and lead to fertility, and the possible interrelationships between these trace elements. Infertile men had higher mean concentrations of plasma copper than those of proven fertility. Concentrations of plasma zinc, erythrocyte zinc, whole blood lead and cadmium, and seminal plasma zinc and copper did not differ significantly between infertile and fertile men. There was a significant positive relationship between sperm density and seminal plasma zinc concentration in the fertile, but not in the infertile, men. Study results confirmed the previously reported association between blood cadmium and smoking and also demonstrated a relationship between cadmium levels and amount smoked, although there were no significant differences in cadmium concentration between fertile and infertile men. (Auth. Abs. Mod.)

84–0725. Steenhoek, A.; Palmen, F.M.L.H.G. **Sustained-Released Theophylline Preparations. A Comparative Study of 467 Patients.** *Respiration* 45(1): 1-15, January-February 1984.

Compliance with a theophylline treatment regimen was better among nonsmokers than among smokers in a comparative evaluation of three sustained-release preparations. Treatment efficacy and therapy compliance were measured in 282 hospital patients (107 smokers) and 185 outpatients (108 smokers). Plasma theophylline concentrations in smokers tended to be lower than in nonsmokers independent of dose or brand of sustained-release preparation. Avoidance of treatment was greater among outpatients (up to 36 percent) compared with hospital patients (less than 5 percent). It is concluded that both smoking and complex treatment schedules increased noncompliance.

84–0726. Stein, R.; Evans, S.; Milner, R.; Rand, C.; Dolovich, J. **Isotopic and Enzymatic IgE Assays in Non-Allergic Subjects.** *Allergy* 38(6): 389-398, August 1983.

In establishing normal serum immunoglobulin E (IgE) concentrations in Canadian Caucasian adults, there was no relationship with age, but among men, levels were significantly higher for smokers than for nonsmokers. Differences were not significant in the female population. A standard isotopic procedure was compared with a new enzymatic counterpart. By the isotopic method, serum IgE concentrations in women and men were comparable from one age group to another with no age-related trend in the seven age groups examined. The median and 95th percentile units, (U)/ml, were 17.5 and 145 for 224 women and 25.5 and 275 for 222 men, respectively. Levels were significantly higher in men. The 95th percentile values are considered the upper limits of normal. The isotopic and enzymatic methods of IgE determination were highly comparable and either is appropriate for routine diagnostic use. (Auth. Abs. Mod.)

84–0727. Stolerman, I. P.; Pratt, J. A.; Garcha, H. S.; Giardini, V.; Kumar, R. **Nicotine Cue in Rats Analysed With Drugs Acting on Cholinergic and 5-Hydroxytryptamine Mechanisms.** *Neuropharmacology* 22(9): 1029-1037, September 1983.

The nicotine discriminative stimulus (cue), used to characterize the underlying receptor mechanisms, was studied in rats trained to discriminate the effects of nicotine in a standard, two-bar operant conditioning procedure with food reinforcement. Mecamylamine blocked both the discriminative effect of nicotine and the reducing effect on the response rate. The block of the discriminative effect could not be overcome by increasing the dose of nicotine, whereas the block of the reducing effect on the response rate could be reversed. Mecamylamine was effective when administered by either the subcutaneous or the intraventricular route, but hexamethonium was inactive by both routes. Mecamylamine, but not hexamethonium, blocked the discriminative effect of the nicotinic-cholinergic agonist, cytisine. Methergoline did not block the discriminative effects of nicotine, even in doses considerably larger than those that blocked the discriminative effects of the 5-hydroxytryptamine (5-HT) agonist, quipazine. Mecamylamine did not block the effects of quipazine. Results are consistent with the view that the nicotine cue is mediated primarily through cholinergic receptors, and that 5-HT mechanisms are not involved. The block of the quipazine cue supports the view that the discriminative effects of this drug are mediated through 5-HT receptors. (Auth. Abs.)

84–0728. Szyba, K.; Lange, A. **Presentation of Benzo(*a*)pyrene to Microsomal Enzymes by Asbestos Fibers in the *Salmonella*/Mammalian Microsome Mutagenicity Test.** *Environmental Health Perspectives* 51: 337-341, September 1983.

Results showing that benzo(*a*)pyrene (BaP) is more readily accessible to the microsomal enzymes when presented as a compound adsorbed onto asbestos particles clarify the amplifying effect of asbestos exposure and cigarette smoking in lung cancer etiology. Amosite, crocidolite, and chrysotile asbestos were equally effective in BaP presentation. Both (1) asbestos fibers without a coating of BaP, and (2) BaP when not dissolved in DMSO lacked any mutagenic effect in the *Salmonella*/mammalian microsome mutagenicity test. (Auth. Abs. Mod.)

84–0729. Tjalve, H.; Castonguay, A. **The *In Vivo* Tissue Disposition and *In Vitro* Target-Tissue Metabolism of the Tobacco-Specific Carcinogen 4-(Methylnitrosamino)-1-(3-Pyridyl)-1-Butanone in Syrian Golden Hamsters.** *Carcinogenesis* 4(10): 1259-1265, 1983.

Results demonstrate that tumorigenesis is correlated with 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) metabolism and binding in target tissues and support the hypothesis that α-carbon hydroxylation is the mechanism of activation of NNK, a tobacco-specific N-nitrosamine. NNK, labeled with ^{14}C in the carbonyl group, was given i.v. or s.c. to Syrian golden hamsters. Whole-body autoradiography indicated that NNK is distributed evenly in the intra- and extracellular tissue water and reaches all tissues. A high level of tissue-bound metabolites occurred in the mucosa of the lungs and the trachea. The liver was the only nontarget tissue that retained detectable tissue-bound metabolites in vivo. Levels of in vitro degradation of NNK to tissue-bound and unbound metabolites were the highest in the nasal mucosa, which, with the lung and the trachea, produced considerable amounts of metabolites that are probably derived from α-carbon hydroxylations of NNK. In the liver, however, the main metabolite was a reduction product of NNK. (Auth. Abs. Mod.)

84–0730. Wilson, P.W.F.; Savage, D. D.; Castelli, W. P.; Garrison, R. J.; Donahue, R. P.; Feinleib, M. **HDL-Cholesterol in a Sample of Black Adults: The Framingham Minority Study.** *Metabolism: Clinical and Experimental* 32(4): 328-332, April 1983.

While smokers among the 100 black adults from Framingham, Massachusetts, smoked more than whites, the difference was not significant for either sex, and the proportion of blacks who smoked did not differ from whites. The black residents were examined and plasma lipids determined. The subjects (45 men, 55 women) ranged in age from 20 to 69; mean age was 42 for both sexes. Mean plasma total cholesterol, high density lipoprotein cholesterol (HDL-C), and triglyceride values for men were 184, 37.2, and 78 mg/dl, respectively; for women, values were 192, 50.4, and 49, respectively. Even adjusting for smoking, alcohol consumption, and obesity, HDL-C levels among the blacks were significantly lower than levels for Framingham white men and women. It is concluded that this black population, which is more educated than previous groups studied (and as active as the white sample), has quite low HDL-C levels that suggest the lipoprotein distributions in this group differ from those previously reported for blacks. (Auth. Abs. Mod.)

84–0731. Witherspoon, L. R.; Shuler, S. E.; Alyea, K.; Husserl, F. E. **Carcinoembryonic Antigen: Assay Following Heat Compared With Perchloric Acid Extraction in Patients With Colon Cancer, Non-Neoplastic Gastrointestinal Diseases, or Chronic Renal Failure.** *Journal of Nuclear Medicine* 24(10): 916-921, October 1983.

Heat inactivation has been proposed as an alternative to perchloric acid (PCA) precipitation for the extraction of carcinoembryonic antigen (CEA) from human plasma. Results using a commercial radioimmunoassay kit (heat inactivation), when compared with results obtained with PCA precipitation, showed that, as in other studies, smoking may elevate the CEA estimate made using heat inactivation. An upper limit for "normal" for the heat-inactivation assay of 3.5 μg/l is based on multiple measurements made in patients (after colon cancer resection) with 2- to 16-year disease-free intervals. The only patients with CEA concentrations >3.5 μg/l were smokers. Adequate sensitivity (1.5 μg CEA/l plasma), satisfactory analytical recovery of CEA added to plasma, and dilutional linearity of samples found to have elevated CEA concentrations, were demonstrated for the heat inactivation assay. Between-assay precision was better with the heat inactivation than with the PCA assay. Although the absolute concentration of CEA estimated after heat inactivation was consistently lower than that estimated after PCA extraction of plasma specimens, there was excellent correlation between results obtained with the two methods in colon cancer patients free of disease, colon cancer patients with residual or recurrent disease, patients with benign gastrointestinal disease, and in patients with chronic renal failure. It is concluded that the heat inactivation assay is an excellent alternative to the PCA assay. (Auth. Abs. Mod.)

84–0732. Woodworth, C. D.; Mossman, B. T.; Craighead, J. E. **Interaction of Asbestos With Metaplastic Squamous Epithelium Developing in Organ Cultures of Hamster Trachea.** *Environmental Health Perspectives* 51: 27-33, September 1983.

Results suggest a mechanism whereby inhaled asbestos and cigarette smoke components could act synergistically in bronchogenic carcinoma induction. The interaction of chrysotile and crocidolite asbestos with cells of the mucociliary and squamous epithelium was studied using both scanning electron microscopy and transmission electron microscopy. Long asbestos fibers were cleared, whereas shorter fibers were phagocytized by cells of the mucociliary epithelium. In contrast, asbestos was phagocytized by superficial squamous cells regardless of fiber length, and fibers penetrated between intercellular junctions in the metaplastic epithelium. Since (1) squamous metaplasia occurs commonly at bronchial bifurcations where appreciable amounts of inhaled materials are deposited by impaction; and (2) metaplastic areas might interrupt the normal mucociliary clearance of materials resulting in their interception and accumulation, a prolonged interaction between inhaled carcinogens and the respiratory epithelium could result. An interaction between asbestos and the underlying layer of basal cells might occur by several mechanisms, including the intercellular penetration of long fibers or by intracellular transport of short fibers by squamous cells. Alternatively, in the normal epithelium, exposure to asbestos results in cytotoxicity and sloughing of ciliated and goblet cells, exposing the underlying basal cells to the fibers. (Auth. Abs. Mod.)

84–0733. Woole, N.; Pittilo, R. M.; Machin, S. J. **Cigarette Smoking and Platelet Adhesion. (Letter).** *Lancet* 2(8358): 1091, November 5, 1983.

A response to several hypotheses concerning increased vascular disease incidence in cigarette smokers (*Lancet* 1(8336): 1248-1250, June 4, 1983; and 2(8350): 627-628, September 10, 1983) suggests that acute exposure of rats to cigarette smoke induces changes in circulating platelets and/or plasma that initially cause increased platelet adherence to an intact endothelial surface. Chronic exposure to smoking may therefore cumulatively result in endothelial cell damage and formation of platelet microthrombi. Relevant observations are described. See also 83-1568 and 84-0398.

84–0734. Wu, K. M.; Martin, W. R. **An Analysis of Nicotinic and Opioid Processes in the Medulla Oblongata and Nucleus Ambiguus of the Dog.** *Journal of Pharmacology and Experimental Therapeutics* 227(2): 302-307, November 1983.

Effects of opioids and nicotine on blood pressure and heart rate were studied by microinjections in the region of nucleus ambiguus of decerebrated dogs. Nicotine (5 μg/0.5 μl) microinjected into the region of nucleus ambiguus produced hypotension accompanied by either bradycardia or tachycardia. Both types of response were antagonized by mecamylamine (10 μg/0.5 μl) but not by naloxone (10 μg/0.5 μl). Fentanyl (5 μg/0.5 μl) and ethylketazocine (10 μg/0.5 μl) decreased blood pressure and heart rate after microinjection into the nucleus ambiguus. The actions of fentanyl were antagonized by naloxone (10 μg/0.5 μl) and N-allylnormetazocine (10 μg/0.5

μl), but not by mecamylamine (10 μg/0.5 μl). Naloxone (10 μg/0.5 μl) administered after ethylketazocine elevated blood pressure and tended to increase heart rate. Naloxone, mecamylamine, or N-allylnormetazocine did not produce significant effect when microinjected alone. It is concluded that, under the present experimental conditions, nicotine and opioid μ, κ but not s receptors are distributed in the region of nucleus ambiguus. No evidence for an interaction between nicotine and opioids in this nucleus was obtained. Furthermore, there was no detectable endogenous opioid or cholinergic nicotinic tone in the nucleus ambiguus of the decerebrated dog. (Auth. Abs.)

84–0735. Zahn, R. K. **Measurement of the Molecular Weight Distributions in Human Muscular Deoxyribonucleic Acid.** *Mechanisms of Ageing and Development* 22(3/4): 355-379, July-August 1983.

Data suggest that high molecular weight deoxyribonucleic acid (DNA) integrity is lost in postmitotic tissue of heavy smokers with increasing age or time of smoking. Highly purified DNA was prepared from human muscle tissue of 328 donors and treated with three different single-strand specific deoxyribonucleases. The molecular weight of treated individual DNA molecules was determined by length measurements. Average DNA molecular weights and their standard deviations were correlated to the age of donors. No significant correlation could be established using seven different mathematical functions. When factor analytical considerations suggested grouping according to extreme smoking habits, two populations with significant double linear correlations of age vs. molecular weight and standard deviation then emerged. (Auth. Abs. Mod.)

See also, **84–0833**

MORTALITY AND MORBIDITY

84–0736. Greitemeyer, M. **Rauchen als Riskantes Gesundheitsverhalten. [Smoking as a Risky Health Behavior.]** *Medizinische Welt* 34(25/26): 713-716, June 24, 1983, German.

The life expectancy of smokers is clearly less than that of nonsmokers; depending on current age, the difference is between 5 and 7 years. Cigarette smoking is a risk factor in the development of coronary heart disease and peripheral vascular disease, and is the main cause of obstructive pulmonary disease and lung cancer. The degree of hazard varies according to the type of tobacco product smoked, daily consumption, age at onset of smoking, and the duration of the habit. Cigarettes are considered more dangerous than pipes, and the earlier, longer, and more one smokes/day, the greater the risk. The term "risky health behavior" is defined and several other examples are given.

84–0737. Higgins, I.T.T.; Glassman, J. H.; Oh, M. S.; Cornell, R. G. **Mortality of Reserve Mining Company Employees in Relation to Taconite Dust Exposure.** *American Journal of Epidemiology* 118(5): 710-719, November 1983.

Analysis of mortality data on 5,751 men, who had worked a year or more for the mining company between 1952 and 1976, suggests that there was no increase in cancer mortality related to taconite exposure; however, a strong relationship was found between smoking habits and mortality from all causes, cardiovascular diseases, and cancer. There were high ratios of standardized mortality to observed deaths for all smokers. Occupational exposures to dust were based on personal samples taken over the past 5 years by the industrial hygiene department of the company. Smoking habits were obtained by mailed questionnaires or telephone interviews. A modified life table method was used to compare death rates of the employees with those expected for white males in Minnesota and with U.S. rates for white males. Results showed that the death rates for all causes were significantly below expectation, while deaths from malignant diseases were marginally below those expected for the State. Exposures to dusts and fiber were low, and there was no relationship between mortality and estimated lifetime dust exposures. The rather unusual high ratio of respiratory cancer in those who smoked tobacco other than cigarettes may be due to the use of a proxy to obtain information on the smoking habits of those who had died. (Auth. Abs. Mod.)

84–0738. Masironi, R. **Smoking and Health: Research Findings in Developing and Developed Countries. Plenary Session IV.** World Health Organization, Geneva, December 1, 1983, 7 pp.

Estimates indicate that annual premature avoidable deaths caused by smoking exceed 500,000 in Europe, 300,000 in the United States, and over 1 million worldwide. Costs that countries must bear relating to medical expenses, production losses, and survivors' pensions resulting from such widespread morbidity and mortality are far higher than the revenue derived from tobacco production. Since 1974, when the World Health Organization warned that the control of cigarette smoking in developed countries would do more to prolong life and prevent disease than any other single action, substantial evidence has shown that the smoking epidemic will have affected the developing world within a decade, before communicable diseases and malnutrition have been controlled. The gap between rich and poor countries will thus be further expanded. Whereas the smoking epidemic is becoming more controlled and smoking trends are declining in the industrialized countries, in developing countries the marketing pressure of the tobacco industry is exerted unchecked. Smoking prevalence rates in developed and developing countries, particularly the increasing number of women who smoke, and related health problems are discussed. (Auth. Abs. Mod.)

See also, **84–0747, 84–0884**

NEOPLASTIC DISEASES

84–0739. Beauchamp, G.; Lassonde, J.; Laurendeau, F.; Leveille, A. **Lung Cancer and Peripheral Vascular Surgery.** *Canadian Journal of Surgery* 26(5): 472-474, September 1983.

Lung cancer, a disease of epidemic proportions, is to some extent preventable; elimination of smoking would certainly reduce its incidence. Conventional therapy has not produced any major advance in this field; the 5-year survival of all patients with lung cancer is still very low. The disease is best managed when detected at an early stage, but results of a Mayo Clinic study of men over age 45, who smoked >20 cigarettes/day, showed no benefit from lung cancer screening of the general population. A few high-risk groups of patients have been identified, such as those treated surgically for laryngeal or pharyngeal tumors, those previously operated on for lung carcinoma, and those who must undergo peripheral vascular surgery. Of 676 patients operated upon for peripheral vascular disease, lung cancer was found in 3.25 percent. The distinction is made between those whose lung cancer was detected at the time of vascular surgery (synchronous) and those whose cancer was found later (metachronous). Every patient scheduled to undergo surgery for peripheral vascular disease should be screened by chest roentgenography and cytologic examination of the sputum. These patients should be followed up closely in the postoperative period. (Auth. Abs.)

84–0740. Binstock, M.; Krakow, D.; Stamler, J.; Reiff, J.; Persky, V.; Liu, K.; Moss, D. **Coffee and Pancreatic Cancer: An Analysis of International Mortality Data.** *American Journal of Epidemiology* 118(5): 630-640, November 1983.

While previous studies have demonstrated a significantly increased pancreatic cancer mortality rate among smokers, only a weak and nonsignificant relationship was found for either total tobacco or cigarette consumption and pancreatic cancer in a survey of 22 countries. The study assessed the relationship of per capita coffee imports and consumption, total dietary fat, saturated fat, cholesterol, tobacco, cigarettes, and national income for 1957-1965 to age-adjusted pancreatic cancer death rates of men and women from 22 countries in 1971-1974. With simple correlation analysis, coffee, total dietary fat, saturated fat, and national income were each significantly correlated with both male and female pancreatic cancer mortality. Bivariate partial correlation coefficients of coffee with pancreatic cancer mortality were significant in 11 of 12 analyses and borderline significant in two-way analyses of variance, controlling for each of the other variables. Saturated fat and pancreatic cancer were also significantly related in univariate analyses. Findings are consistent with the hypothesis that coffee and dietary lipids are involved in the etiology of pancreatic cancer. (Auth. Abs. Mod.)

84–0741. Broitman, S. A.; Vitale, J. J.; Gottlieb, L. S. **Ethanolic Beverage Consumption, Cigarette Smoking, Nutritional Status, and Digestive Tract Cancers.** *Seminars in Oncology* 10(3): 322-329, September 1983.

The apparent synergistic relationship between alcohol consumption and smoking in digestive tract cancer development is discussed in a detailed review of the role of alcohol consumption in cancer etiology. The relationship has been observed in individuals who consume ≥45 ml alcohol/day. While conflicting data from studies make it difficult to separate the processes that contribute to the suggested synergism, the instance of cancers of the upper respiratory and alimentary tract that occur independently of alcohol or tobacco consumption might imply that the addition of these factors might aid and abet a primary process, i.e., impaired nutritional status, in oncogenesis at these sites. At present, 11 percent of the adult population (16.7 percent of all users of alcohol) are classified as alcohol abusers, consuming approximately 50 percent of the alcoholic beverages produced and sold legally in the United States. The effect of the high caloric intake from alcohol in abusers (29.1 percent and 40.3 percent of daily intake in males and females, respectively) on nutritional status and the resultant carcinogenesis relationship is also described.

84–0742. Cartwright, R. A.; Adib, R.; Appleyard, I.; Glashan, R. W.; Gray, B.; Hamilton-Stewart, P. A.; Robinson, M.; Barham-Hall, D. **Cigarette Smoking and Bladder Cancer: An Epidemiological Inquiry in West Yorkshire.** *Journal of Epidemiology and Community Health* 37(4): 256-263, December 1983.

Results of a case—control study (1,259 cases, 1,981 controls) show, for the first time in the United Kingdom, a positive but weak dose—response relationship between cigarette smoking and bladder cancer. Data indicate a complex set of relationships when quantity smoked and period of smoking are considered: a dose—response effect is weakly demonstrated when 10 to 20 cigarettes/day are smoked, but no effect is found in those who smoked the greatest quantity for the longest periods. While results indicate that the risks increase according to different length of time smoked, there is no clear ordinal sequence. There appears to be no risk for those who smoke only filter cigarettes. Results are contrasted with similar studies, and significance of the observations is discussed. (Auth. Abs. Mod.)

84–0743. Castot, A.; Efthymiou, M.-L. **Cancers du Tabagisme. [Cancer and Passive Smoking.]** *Semaine des Hopitaux* 59(27/28): 2028-2030, July 7, 1983, French.

The proximity of smokers exposes nonsmokers to the inhalation of tobacco smoke (passive smoking). Assays of blood nicotine concentrations or urinary thiocyanate concentrations have established that being near an individual who smokes approximately 10 cigarettes is equivalent to smoking one cigarette. The consequences of such exposure are not negligible; two studies (one Japanese, one Greek) have shown that the risk of lung cancer in wives of smokers is twice that of nonsmoking women married to nonsmokers. (Various American studies are more reassuring.) The risk of cancer also exists in the offspring of mothers who smoked during pregnancy, a risk confirmed by experiments with hamster fetuses. (Auth. Abs.)

84–0744. Chailleux, E. **Cancers Broncho-Pulmonaires et Facteurs Professionnels. [Broncho-Pulmonary Cancer and Occupational Factors.]** *Archives des Maladies Professionnelles de Medecine du Travail et de Securite Sociale* 43(8): 687-689, 1982, French.

General knowledge of the physico-chemical basis of carcinogenesis of bronchopulmonary cancer, and the specific physical and chemical carcinogenic agents, are reviewed. The synergistic carcinogenic action of smoking and ionizing radiation or asbestos exposure is stressed. The mechanism of chemical carcinogenesis of the polycyclic hydrocarbons from cigarette smoke is explained.

84–0745. Cumming, G. **Inhaling and Lung Cancer. (Letter).** *British Medical Journal* 288(6411): 145-146, January 14, 1984.

Conclusions of a recent paper (*British Medical Journal* 287(6401): 1273-1275, October 29, 1983) are disputed on the grounds that, in the lungs, carbon monoxide (CO) behaves as a gas and smoke as particulate matter. Machine-smoked CO yield values ignore dwell time in the lungs as a determinant. Thus, CO levels cannot be used as an index of particle deposition or to explain why deep inhalers of cigarette smoke appear to have fewer carcinomas than light inhalers. See also 84-0456.

84–0746. Curnen, M.G.M. **Lung Cancer in Connecticut Women. (Letter).** *Lancet* 2(8355): 906-907, October 15, 1983.

A 1977 prediction that lung cancer mortality among women would surpass breast cancer mortality has come true. Data from the Connecticut Tumor Registry show that between 1972 and 1981, the number of female lung cancer cases increased by 50 percent, while male lung cancer cases increased by only 15 percent. Between 1955 and 1966, smoking among women increased by 17.2 percent, while decreasing 8.4 percent in men. In 1966, 43.2 percent of women and 58.9 percent of men aged 25 to 34 years were smokers. Recent survey data now indicate that, by the age of 17 to 19, smoking prevalence in females exceeds that of males. Although by 1980 smoking prevalence in women had declined 5 percent from 1966 levels, the number of women consuming >25 cigarettes/day had increased.

84–0747. Davis, D. L.; Bridbord, K.; Schneiderman, M. **Cancer Prevention: Assessing Causes, Exposures, and Recent Trends in Mortality for U.S. Males, 1968-1978.** *International Journal of Health Services* 13(3): 337-372, 1983.

The smoking issue is considered in a review of recent trends in chemical production and cancer mortality rates. The methodological problems of estimating cancer causes and risks, with smoking as a confounding factor, are discussed. Assessing exposure to carcinogens is accomplished by documenting countervailing trends in chemical production during the past two decades, noting the doubling of some synthetic organic human carcinogens and the leveling off of some heavy metal carcinogens. Recent increases in site-specific causes of cancer mortality for men old enough to have developed workplace cancers (ages 35 to 84) are detailed, considering those cancers that have been linked with exposures to toxic chemicals and to cigarette smoking. A sharp reduction in cancer mortality for those under age 45 more than offsets increases in some cancers for those ages 45 to 65. Men aged 55 to 84 have experienced major increases in mortality for certain cancers plausibly associated with occupational exposures, including cancers of the brain, lung, and multiple myeloma. These older age groups have potentially sustained longer workplace exposures to carcinogens, some of which have 25-year or greater latencies. (Auth. Abs. Mod.)

84–0748. Devesa, S. S.; Diamond, E. L. **Socioeconomic and Racial Differences in Lung Cancer Incidence.** *American Journal of Epidemiology* 118(6): 818-831, December 1983.

Information regarding smoking habits was not routinely gathered during the Third National Cancer Survey, the basis for this report, and thus it was not possible to directly study the impact of smoking and its interplay with socioeconomic status. However, the observed lung cancer rates stratified by income and education levels were generally consistent with those predictable from studies of smoking habits in other population samples. However, smoking may not explain all the observed variations in risk by social class. The association of lung cancer incidence with income and education, and the effect of adjustment for socioeconomic distribution on black—white differences in lung cancer rates were evaluated using data from the Third National Cancer Survey. Included were 20,868 cases of lung cancer (18,514 among whites and 2,354 among blacks) diagnosed among metropolitan residents during 1969-1971. Median family income and median years of education by census tract of residence were used to indicate socioeconomic group. Strong significant inverse trends between lung cancer incidence and both income and education were apparent among white and black males. The effect of income exceeded that of education. Lung cancer rates among black males compared with white males were significantly higher ($p<0.001$) before socioeconomic adjustment, nonsignificantly higher after adjustment for education, and nonsignificantly lower after adjustment for income. Strong trends in risk with income or education were not observed for lung cancer among females of either race. (Auth. Abs. Mod.)

84–0749. Dubrow, R.; Wegman, D. H. **Setting Priorities for Occupational Cancer Research and Control: Synthesis of the Results of Occupational Disease Surveillance Studies.** *Journal of the National Cancer Institute* 71(6): 1123-1142, December 1983.

Most of the associations between occupation and cancer for which there are reasonable exposure hypotheses involve cancer sites associated with cigarette smoking (respiratory tract, oral cavity, esophagus, larynx, and bladder), and thus may be traced to occupational exposure, smoking, or both. In two large studies that considered smoking histories, most associations between occupation and lung cancer remained unchanged when controlled for smoking. The results of 12 major occupational disease surveillance studies were synthesized to (1) identify occupations with potentially high cancer risk by combining the results of studies; (2) develop a quantitative methodology for accomplishing the first objective; and (3) make recommendations concerning priorities for occupational cancer research and control based on the results of this analysis and other available epidemiologic, industrial hygiene, toxicologic, and employment data. It is suggested that the first priority be the investigation and control of occupational exposure to asbestos, particularly in the automobile repair and construction industries. Of the 34 occupational groups found to be at high risk for lung cancer in this analysis, 18 have potential asbestos exposure. The second priority is research into the consistent excess of lung cancer among motor vehicle drivers, which may be due to occupational exposure to diesel and gasoline engine exhaust, to cigarette smoking, or to both. (Auth. Abs. Mod.)

84–0750. Flanders, W. D.; Cann, C. I.; Rothman, K. J.; Fried, M. P. **Work-Related Risk Factors for Laryngeal Cancer.** *American Journal of Epidemiology* 119(1): 23-32, January 1984.

Data from a case—control study on employment-related risk factors for laryngeal cancer suggest that employment is associated with increased risk that is not entirely the result of heavier smoking or drinking by workers in a particular occupation. All persons with newly diagnosed squamous cell carcinoma of the larynx treated in the area hospitals in Richmond County, Georgia, and contingent counties were identified. Potential controls were selected from patients admitted to these hospitals during the same time period as the cases, excluding those patients admitted for cancer, lung disease, or employment-related disease. Eighty-five control subjects were individually matched to 42 cases for sex, age, area of residence, and smoking and alcohol-drinking history. Subjects were matched by alcohol and tobacco consumption categories; 64 percent of both cases and controls had a history of over 40 pack-years of smoking. From lifetime employment histories, laryngeal cancer rate ratios were estimated comparing the incidence rate among subjects who had ever worked in an occupation with the incidence rate among subjects who had never worked in that occupation. Among the 10 occupations in which at least 15 subjects had worked, rate ratio estimates were found to be above 3.0 for farmers, textile processors who separated, filtered, or dried textile fibers, and for all laborers and maintenance personnel. (Auth. Abs. Mod.)

84–0751. Fraumeni, J. F., Jr. **The Face of Cancer in the United States.** *Hospital Practice* 18(12): 81-96, December 1983.

Geographic analysis by county of cancer mortality data in the United States reveals a surprising variety of patterns. Mortality from lung cancer among white males is high in metropolitan centers of the Northeast, consistent with higher smoking rates in urban vs. rural populations. However, the highest rates for lung cancer among white males are clustered in coastal areas of the South, inversely related to socioeconomic status but related to chemical and petroleum industries nationwide and with the paper and pulp manufacturing industry in the East and South. A case—control study in coastal Georgia revealed a synergistic interaction between shipyard employment and tobacco smoking, with a risk 20 times higher among shipyard workers who were heavy smokers, compared with nonsmoking, nonshipyard workers. The excess risk in shipyard workers is probably asbestos-associated, but no excess cases of mesothelioma were found. Shipworkers in the Tidewater area of Virginia did show an excess of mesothelioma, with a risk of 18.1 for asbestos handlers. The highest lung cancer rate in the United States was found in Duval County (Jacksonville), Florida; increased risks of lung cancer (40 to 50 percent) were associated with shipbuilding, construction, and lumber or woodworking industries. Many workers reported asbestos exposure, but other carcinogenic hazards may be involved. A nasal cancer study in North Carolina and Virginia furniture workers revealed excess risk associated with smoking and wood-related occupations. Wood dust may act as a promoter. Changing patterns in lung cancer death rates (a rise in nonwhite male deaths, now 50 percent higher than in white males born around 1915) seem to reflect changes in smoking practices. More whites than blacks have given up smoking. Recent studies link lung cancer risk to reduced vitamin A intake, primarily among smokers. Excess deaths among females in the Southeast (90 percent excess in rural areas) due to oral and pharyngeal cancer were found to result from snuff-dipping. Excess mortaity among black males in Washington, D.C., from esophageal cancer was found to be dose-dependent on alcohol consumption; the alcohol association probably masked the smoking-associated risk. Nutritional status was also strongly associated with esophageal cancer; generally poor nutrition, along with alcohol consumption, probaby contributes to the elevated rates among blacks. Clustering, occupational, and nutritional factors for prostate, breast, colon, stomach, kidney, pancreatic, hematopoietic, and lymphatic cancers are also discussed. Cigarette smokers have a 60 percent excess risk of renal adenocarcinoma, and a six- to sevenfold excess of cancer of the renal pelvis, reaching 11-fold among heavy smokers, has been demonstrated. Mapping of anatomic site-specific cancer death rates by counties or groups of counties yields a rich source of clues about environmental factors, which can then be confirmed by epidemiological investigations.

84–0752. Gillis, T. M.; Incze, J.; Strong, M. S.; Vaughan, C. W.; Simpson, G. T. **Natural History and Management of Keratosis, Atypia, Carcinoma In Situ, and Microinvasive Cancer of the Larynx.** *American Journal of Surgery* 146: 512-516, October 1983.

In a retrospective study of 42 patients (38 men, 4 women), with leukoplakia or erythroplakia (or both) of the vocal cords, it was found that cessation of smoking by 15 of the original 39 smokers offered little protection against the development of new lesions. The visible dysplastic lesions of the larynx are apparently an expression of the continuum of epithelial breakdown that occurs in response to prolonged exposure to carcinogens (most often tobacco tar). Carcinoma in situ or microinvasive cancer was present initially or eventually developed during followup in 33 patients. Keratosis, atypia, carcinoma in situ, and microinvasive cancer occurring as white or red patches on the vocal cords are part of the diathesis of cancer of the aerodigestive tract and represent a sequential continuum. Excisional biopsy is the preferred treatment for identification and potential cure of the lesion. Since cessation of smoking does not remove the potential for progression of the disease, all patients must be followed indefinitely. (Auth. Abs. Mod.)

84–0753. Gonvers, J.-J. **Tabagisme et Gastro-Enterologie. [Smoking and the Gastrointestinal Tract.]** *Therapeutische Umschau* 40(2): 139-142, 1983, French.

Several studies have shown increased mortality from esophageal cancer among smokers. Male smokers in one study had a mortality rate of 10/100,000, compared with 4/100,000 for nonsmokers. Risk, which is proportional to amount smoked, is increased by alcohol consumption. Pancreatic cancer is increasing in both sexes, and in Western countries prevelence is estimated at 9.5/100,000 inhabitants. One study showed that smokers developed pancreatic cancer 10 to 15 years earlier than nonsmokers, while another established a relationship with the number of cigarettes smoked. Three mechanisms are suggested to explain the carcinogenic effect of tobacco on the pancreas: (1) inhaled carcinogens are transported to the pancreas in the bloodstream; (2) carcinogens excreted in bile reach the pancreas via reflux through the Wirsung channel; and (3) smoke-induced increases in blood lipids may promote pancreatic carcinogenesis. Smoking is associated with the incidence, slow healing, relapse, and increased mortality from ulcerous disease; stopping smoking promotes healing of gastric ulcers. Also, tobacco consumption may influence alcohol-related risk of stomach cancer and esophageal reflux, although the mechanisms are not known.

84–0754. Grossenbacher, R. **Rauchen und Seine Folgen in der Otorhinolaryngologie. [The Effects of Tobacco Smoking in Otorhinolaryngology.]** *Therapeutische Umschau* 40(2): 125-132, 1983, German.

The relationship of smoking, which affects all the mucous membranes of the upper respiratory and digestive tract, with carcinoma of the mouth, larynx, oropharynx, and hypopharynx, and treatment of these cancers, are reviewed. One study of 300 cases of preneoplastic or cancerous lesions of the oral cavity found that 15 percent also had lesions in the larynx. Smoking-related neoplasms account for three-fourths of the cancers at these sites, most appearing as basal cell carcinoma. Cigarette-only smokers have a relative risk of 15.8, those smoking cigarettes, cigars, and pipes a risk of 10.0, while cigar-only smokers have a relative risk of 3.9. Concurrent alcohol consumption further increases smoking risks. Over the past 20 years, cigar and pipe smoking have declined, while cigarette consumption has increased. Oral carcinoma also decreased, possibly implying a greater connection to cigars and pipes than to cigarettes. The importance of early diagnosis by dentists and physicians, and antismoking education among patients, is emphasized.

84–0755. Grufferman, S.; Delzell, E. S.; Maile, M. C.; Michalopoulos, G. **Parents' Cigarette Smoking and Childhood Cancer.** *Medical Hypotheses* 12(1): 17-20, September 1983.

A recent study of childhood rhabdomyosarcoma (RMS) (*Journal of the National Cancer Institute* 68(1): 107-113, January 1982) found an increase in the risk of RMS among children whose fathers smoked cigarettes; however, there was no association between RMS and mothers' smoking. It is hypothesized that differential germ cell damage from cigarette smoking may be responsible, and that such a risk is greater for men than for women. The increased susceptibility for male germ cells may be due to the number and timing of meiotic and mitotic cell divisions. In males, germ cells undergo large numbers of meiotic and mitotic divisions throughout the reproductive years. In females, however, generally only one oocyte matures and completes meiosis each month of the reproductive years. Thus, there are very large male-female differences in the number of rapidly dividing germ cells during the reproductive years, and it is rapidly proliferating cells that are most susceptible to genetic damage. Thus, fathers' environmental exposures before conception of children may play a role in the etiology of childhood cancer. (Auth. Abs. Mod.) See also 82-0673.

84–0756. Hecht, S. S.; Adams, J. D.; Numoto, S.; Hoffmann, D. **Induction of Respiratory Tract Tumors in Syrian Golden Hamsters by a Single Dose of 4-(Methylnitrosamino)-1-(3-Pyridyl)-1-Butanone (NNK) and the Effect of Smoke Inhalation.** *Carcinogenesis* 4(10): 1287-1290, 1983.

Results demonstrated that even a single dose of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) can induce respiratory tract tumors in Syrian golden hamsters. Smoke inhalation, however, did not result in an increase in respiratory tract tumor incidence in most of the NNK-treated groups. Four groups of 10 male and 10 female Syrian golden hamsters were given single s.c. injections of either 0.3 ml of trioctanoin or of 0.3 ml of trioctanoin containing either 1.0 mg, 3.3 mg, or 10.0 mg of NNK, a tobacco-specific carcinogen. Hamsters were then exposed to cigarette smoke for the next 72 weeks. Four control groups received the same injections of NNK or trioctanoin, but were treated by sham smoking. All groups treated with NNK had tumors of either the lung, nasal mucosa, and/or trachea. These tumors were not observed in hamsters injected with trioctanoin. (Auth. Abs. Mod.)

84–0757. Heller, W.-D. **Lung Cancer and Passive Smoking. (Letter).** *Lancet* 2(8362): 1309, December 3, 1983.

Conclusions and histological evidence of a Greek hospital study, which implicated passive smoking as being a lung cancer factor (*Lancet* 2(8351): 677-678, September 17, 1983), are questioned, as is the use of cases and controls from different hospitals. It is suggested that the latest data, compared with an earlier (1981) report, do not show (1) as clear an association between a woman's lung cancer risk and her husband's smoking habits, or (2) the dose—response relationship previously seen. The author further states that, while relative risks reported in the 1981 paper agreed exactly with the appropriate cross-product ratios calculated from the numbers of cases and controls in the relevant category for husbands' smoking, current data on relative risks, despite apparently identical methods, clearly disagree. See also 84-0122.

84–0758. Heuch, I.; Kvale, G.; Jacobsen, B. K.; Bjelke, E. **Use of Alcohol, Tobacco and Coffee, and Risk of Pancreatic Cancer.** *British Journal of Cancer* 48(5): 637-643, November 1983.

Results of a Norwegian prospective study (n = 16,713) clearly associated 63 pancreatic cancer cases with tobacco chewing or snuff-dipping (bordering on statistical significance), and indicated a somewhat weaker positive association with cigarette smoking. Data indicated a relative risk (RR) of 2.04 for male smokers (≥ 10 cigarettes/day) compared with nonsmokers. Associations were assessed using techniques for stratified logistic regression. Of potential risk factors considered, alcohol consumption showed the strongest positive association, with an estimated RR of 5.4 for frequent drinkers compared with nondrinkers ($p < 0.001$). No association was established for pipe smoking or coffee drinking. (Auth. Abs. Mod.)

84–0759. Hirayama, T. **Passive Smoking and Lung Cancer: Consistency of Association. (Letter).** *Lancet* 2(8364): 1425-1426, December 17, 1983.

Results of two recent reports associating passive smoking with lung cancer (*Lancet* 2(8350): 595-597, September 10, 1983; and *Lancet* 2(8351): 677-678, September 17, 1983) are externally consistent with those of a large scale cohort study (122,261 men and 142,857 women, aged 40 years or more) in Japan. Of 429 women who died from lung cancer during 16 years of followup (1966 to 1981), 303 had been nonsmokers; 200 of these deaths occurred among 91,540 nonsmoking married women whose husbands' smoking habits were known. Standardized mortality ratios of lung cancer in nonsmoking women were 1.00, 1.36, 1.42, 1.58, and 1.91 when husbands were nonsmokers, ex-smokers, and daily smokers of < 15, 15 to 19, and > 19 cigarettes/day, respectively. Based on extent of husband's smoking, trends of increased risk among nonsmoking women were similar for different age groups, occupational groups, and duration of observation. Smoking by the husband was the only characteristic found to increase the risk of lung cancer in nonsmoking wives. Without regard to the level of consumption, the lung cancer risk was increased 60 percent. A significantly increased risk of cancer of the paranasal sinuses in nonsmoking wives, related to the amount of husbands' smoking, was also observed. See also 84-0122 and 84-0434.

84–0760. Kimizuka, G.; Hayashi, Y. **Extraction of Ferruginous Bodies From Lung Tissue Obtained at Surgery and Autopsy.** *Acta Pathologica Japonica* 33(4): 715-724, July 1983.

Evidence suggests a relationship between lung cancer and asbestos exposure with smoking. Ferruginous bodies were extracted quantitatively from 508 patients and divided morphologically into four types, two from asbestos and two (rarely found) from carbon and silicate. Cases with asbestos bodies above 100/5 g of wet lung totaled 36 of 242 lung cancer patients (14.8 percent, all smokers) and 15 of 266 patients with other diseases (5.6 percent). Nine of 14 cases with bodies above 300/5 g of wet lung were lung cancer patients and heavy smokers (> 20 cigarettes/day). The remaining five patients included two heavy smokers with renal transplantation and double stomach carcinomas, one moderate smoker (10 to 20 cigarettes/day), one light smoker (eight cigarettes/day), and a nonsmoker.

84–0761. Knoth, A.; Bohn, H.; Schmidt, F. **Passive Smoking as a Causal Factor of Bronchial Carcinoma in Female Nonsmokers.** Complete English translation of **"Passivrauchen**

als Lungenkrebs-Ursache bei Nichtraucherinnen." *Medizinische Klinik* 78(2): 66-69, February 4, 1983.

In a study of 792 patients of both sexes with bronchogenic carcinoma in the Mannheim-Ludwigshaten-Heidelberg region, there were 59 female patients, 39 of them nonsmokers. Of the nonsmoking female patients, 61.5 percent had lived with smokers, triple the number anticipated based on the smoking habits of men in their respective age brackets. Neither indications of occupational exposure nor hereditary factors accounted for the excess. Hence, in retrospect, passive smoking appears to be the most obvious interpretation of the high percentage of nonsmokers among the female patients with bronchogenic carcinoma. In addition, the percentage of squamous cell and small cell carcinoma, the typical smokers' carcinomas, among the nonsmoking wives (66.6 percent) was not significantly lower than that among the females who smoked (80 percent).

84–0762. Lodovici, M.; Dolara, P.; Caderni, G.; Carini, M.; Costantini, A.; Selli, C.; Barbagli, G.; Calzolai, A. **The Effect of Cigarette Smoke on Aryl-Hydrocarbon Hydroxylase (AHH) Activity of the Human Kidney.** *European Journal of Cancer and Clinical Oncology* 19(11): 1565-1568, November 1983.

Aryl hydrocarbon hydroxylase (AHH) activity roughly correlated with amount smoked in cancer and noncancer cases ($p<0.05$). Smoking significantly increased AHH activity in hydronephrotic kidneys, but not in cancer kidneys, probably due to the higher baseline values and scatter in the cancer group. When cancer and noncancer cases were pooled, nonsmokers had a mean value of AHH activity of 9.8 ± 7.8, and smokers had a value of 16.3 ± 6.8 ($p<0.05$). It is concluded that the effect of smoking and not the presence of kidney cancer seems to affect AHH activity levels. Thus, an increased capability of activating procarcinogens is unlikely to be a major cofactor in kidney cancer induction. (Auth. Abs. Mod.)

84–0763. Mommsen, S.; Aagaard, J. **A Case-Control Study of Bladder-Cancer.** *Danish Medical Bulletin* 30(6): 427-432, November 1983.

In a case—control study of 212 bladder cancer patients (165 men, 47 women) and 259 controls (matched for age and geographic area), a significantly increased bladder cancer risk was associated with all types of smoking. Among patients, 134 were cigarette smokers, 104 smoked cigarillos, and 39 chewed tobacco; 193 also consumed alcohol. Multivariate logistic analysis showed that cigarettes constituted the highest independent relative risk (RR) in age groups 40 to 59 and 60 to 69 years, and cigarillos in age groups 40 to 59 and 70 to 89 years. RR, 2.09 times greater for cigarette smokers compared with nonsmokers, more than doubled to 4.57 for smokers of both cigarettes and cigarillos. While cigarette or cigarillo smoking increased RR of bladder cancer in both men and women, the most important determinants were cigarette smoking for men and cigarillo smoking among women. Also, tobacco chewing and previous venereal disease among men, and saccharin consumption among women were strongly associated. Increased RR for bladder cancer was also associated with industrial occupations; work with kerosene, asphalt, oil, gasoline, and chemical materials; and alcohol consumption.

84–0764. Morris, D. L.; Borhani, N. O.; Fitzsimons, E.; Hardy, R. J.; Hawkins, C. M.; Kraus, J. F.; Labarthe, D. R.; Mastbaum, L.; Payne, G. H. **Serum Cholesterol and Cancer in the Hypertension Detection and Follow-up Program.** *Cancer* 52(9): 1754-1759, November 1, 1983.

Smoking, age, percent of ideal weight, and serum cholesterol were all statistically significant variables in an investigation of the relationship between serum cholesterol and cancer incidence in the population of the Hypertension Detection and Follow-up Program. During the 5 years of followup, 286 new cancer cases were documented among the 10,940 participants. Overall, age-adjusted cancer incidence rates, computed by baseline serum cholesterol quartiles, showed a small, but statistically significant, inverse relationship between serum cholesterol and cancer incidence. No evidence suggested that the observed relationship was primarily due to confounding by other cancer risk factors, association of low serum cholesterol with incipient but undiagnosed cancer, or problems of competing risks. However, the relationship is weak and a causal interpretation of these immediate results cannot be argued persuasively. Examinations of specific cancer sites and factors related to serum cholesterol are suggested as important lines of research toward clarification of the complex relationships observed. (Auth. Abs. Mod.)

84–0765. Newell, G. R. **Lifestyles and Cancer Prevention.** *Progress in Clinical and Biological Research* 130: 55-66, 1983.

Two approaches are necessary for cancer prevention: research, and the actions of government, society, and the individual on the knowledge provided by research. However, knowledge that tobacco products cause 35 to 40 percent of cancer has not led to prevention of these cancers, and even if all smoking abruptly ceased, lung cancer would still occur into the next century. Cancer prevention will eventually be implemented by government, or by individuals exercising personal lifestyle choices, although neither has been successful with regard to cigarette manufacture, subsidy, promotion, sales, or consumption. Thus, the ultimate instrument for cancer prevention will be behavior modification, whether in the individual, environment, or medical care system, and success will be measured in small increments.

84–0766. Pai, S. B.; Steele, V. E.; Nettesheim, P. **Neoplastic Transformation of Primary Tracheal Epithelial Cell Cultures.** *Carcinogenesis* 4(4): 369-374, 1983.

Unrestricted cell replication appears to be an early key event in carcinogen-exposed epithelial cell populations, preceding neoplastic transformation. Primary cultures of rat tracheal epithelial cells were treated with the chemical carcinogen N-methyl-N'-nitro-N-nitrosoguanidine (MNNG) to quantitatively study the early events of carcinogenesis. MNNG did not decrease colony forming efficiency (CFE) at a concentration of 0.1 μg/ml, but 1.0 μg/ml reduced the CFE by 75 percent. For transformation studies, primary cell cultures received single exposures to MNNG (0.1 to 0.6 μg/ml) or multiple exposures to 0.1 μg/ml of MNNG for 3 hours between days 1 and 17. In carcinogen-exposed cultures, morphologically altered foci appeared on day 18, recognizable by high cell density. Transformation frequencies between 1 and 8 percent were observed depending on MNNG concentration. Cultures containing altered foci continued to grow during the third and fourth week, when control cultures had ceased to proliferate, and exfoliated from the dish. Over 40 percent of the cultures that received multiple exposures to MNNG acquired cell line status and could be subcultured $\geq$20 times. None of the 30 control cultures became cell lines. Also, 70 percent of MNNG-exposed cell lines showed the anchorage independent growth phenotype at passage 20 as judged by growth in agarose. Of 10 cultures exposed either 6 or 8 times to MNNG, 4 formed invasive squamous cell carcinomas at passage 20 upon inoculation into nude mice. (Auth. Abs. Mod.)

84–0767. Quintero Gonzalez, J. **Cancer Oral en Fumadores. Estudio Retrospectivo de 672 Casos. [Oral Cancer in Smokers. Retrospective Study of 672 Cases.]** *Boletin de Investigaciones* 3(7): 6-9, July 1981, Spanish.

Reverse smoking was practiced by 202 oral cancer patients (30 percent), predominantly by women and most often affecting the hard palate. Of 52,685 cancers diagnosed in the Department of Pathology of the University of Cartagena (Colombia), 672 cases were squamous cell carcinomas of the oral cavity (43 percent men and 57 percent women); 95 percent were over age 40. The most frequent site was the palate (35 percent), followed by the tongue (26.5 percent) and the floor of the mouth (10 percent). The frequency of palatal cancer was over twice as high in women (37 percent) as in men (17.4 percent). Twelve of 187 males and 16 of 385 women were smokers, and an additional 37 men and 165 women were reverse smokers. Frank metastases to the regional nodes were found in 27.5 percent (usually the reason for seeking medical attention). The primary site was not discovered until after clinical examination. Very few cases were detected early (only two cases of cancer in situ were found). Reverse smoking, associated with cancer of the palate, is widely practiced in the Atlantic coastal region of Colombia, especially by laundresses. It is recommended that public promotional programs be developed to facilitate early diagnosis and treatment.

84–0768. Quintero Gonzalez, J. **Registro de Carcinoma Escamocelular en los Departamentos de Cordoba y Sucre. [Register of Squamous Cell Carcinoma in the Provinces of Cordoba and Sucre.]** Colombia, Universidad de Cartagena, 1983, 11 pp., Spanish.

A retrospective study of 329 oral cancer cases in two Colombian provinces revealed that 82 percent of female patients from Sucre and 65 percent from Cordoba were reverse smokers. Fewer males practiced reverse smoking (39 percent and 16 percent, respectively); overall, half the patients were reverse smokers. Of the 207 cases from Cordoba (97 males, 110 females) and 122 from Sucre (57 males, 67 females), the palate was the most frequent cancer site (36 percent in Cordoba and 47 percent in Sucre), and the tongue the next most frequent (27 and 21 percent, respectively). Among the reverse smokers, over 60 percent of all cancers were palatal. Almost all the patients were country people of low socioeconomic status. Characteristically, they came to treatment with advanced disease and poor prognosis. Better health measures are needed to lower the prevalence of these cancers.

84–0769. Quintero Gonzalez, J.; Serrano Serrano, R. **Registro de Carcinoma Escamocelular de Cavidad Oral en Bolivar. [Register of Squamous Cell Carcinoma of the Oral Cavity in Bolivar.]** *Ciencia Tecnologia y Educacion, Universidad de Cartagena* pp. 76-91, October 1981, Spanish.

Among 135 histologically confirmed patients (69 men, 65 women) with squamous cell oral carcinoma in the northwest region of Colombia, 43 smoked "normally," 41 were reverse smokers, and 3 were nonsmokers (no smoking information was available for 48). No patient was under age 30, and 76 percent were over age 60. The most common cancer sites were the palate (42 cases) and tongue (37 cases, 24 at the base of the tongue). The patients were rural people of low socioeconomic status in low-salaried occupations: 30.4 percent were farm workers, 15.5 percent domestics, 23 percent housewives, 3 percent laborers, 3 percent day workers, and 3 percent unemployed. Most came to treatment in late stages of disease; at diagnosis, 51.9 percent had invasive tumors, 13 percent had metastases, and 10.4 percent had both. Only 10.4 percent were diagnosed in dental clinics, the rest by medical professionals, reflecting the ignorance of dental care among the rural poor.

84–0770. Rosenberg, L.; Schwingl, P. J.; Kaufman, D. W.; Miller, D. R.; Helmrich, S. P.; Stolley, P. D.; Schottenfeld, D.; Shapiro, S. **Breast Cancer and Cigarette Smoking.** *New England Journal of Medicine* 310(2): 92-94, January 12, 1984.

Data from 2,160 women with diagnosed breast cancer (33 percent smokers, 23 percent ex-smokers, 42 percent nonsmokers, 2 percent unknown category) do not support the hypothesis that smoking may reduce breast cancer incidence by as much as 20 percent. To evaluate the relationship between breast cancer and smoking, patients were compared with 717 controls who had been admitted for cancers of the ovary, colon or rectum, malignant melanoma, or lymphoreticular cancers. Smoking habits were similar to the breast cancer patients. Compared with women who had never smoked, the estimated relative risk of breast cancer was 1.1 for current smokers of any amount (95 percent confidence interval, 0.9 to 1.3), and 1.0 (0.8 to 1.3) for heavy smokers (≥ 15 cigarettes/day). Allowance for all identified potential confounding factors did not materially alter the results. There was no indication that age of commencement of smoking was related to the risk, nor was there evidence of an effect of smoking within the categories of age at first pregnancy or age at menopause. (Auth. Abs. Mod.)

84–0771. Seitz, H. K.; Czygan, P.; Kommerell, B. **Alkohol und Karzinogenese. [Ethanol and Carcinogenesis.]** *Leber Magen Darm* 12(3): 95-107, 1982, German.

Alcohol enhances carcinogenesis induced by other compounds: smoking or chewing tobacco concurrently with alcohol consumption has a synergistic effect on carcinogenesis in the upper digestive and respiratory tract. While alcohol consumption and smoking increase cancer risk by factors of 18 and 5.1, respectively, used together, the synergistic risk increases to 44.4. Similar statistics apply to carcinoma of the larynx and esophagus. Alcoholics who also smoke have 6 to 15 times the risk of developing oral carcinoma than nonsmokers and nondrinkers. Women who smoke and drink heavily develop tongue and buccal tumors approximately 15 years earlier than women who abstain. Ethanol may influence both the initial and promotional phases of carcinogenesis. Ethanol enhances activation of a number of procarcinogens in the small intestine, liver, and lung. A local effect may be responsible for carcinogenesis of the upper gastrointestinal tract; ethanol is known to influence gastrointestinal regenerative, secretory, motility and microbial patterns. Alcoholic beverages contain carcinogens; additionally, alcohol-induced changes in hormone, vitamin and mineral metabolism, and the immune system may contribute to carcinogenesis.

84–0772. Spitz, M. R.; Batsakis, J. G. **Major Salivary Gland Carcinoma. Descriptive Epidemiology and Survival of 498 Patients.** *Archives of Otolaryngology* 110(1): 45-49, January 1984.

Although case—comparison analysis failed to show a significant excess of tobacco users among patients with histopathologically confirmed carcinoma of the major salivary glands, 237 of the 498 patients smoked, dipped snuff, or chewed tobacco. Of 30 epidermoid carcinoma patients, 23 were tobacco users, as were 59 of 146 patients with mucoepidermoid carcinoma, 50 of 102 adenocarcinoma patients, and 46 of 100 with adenoid cystic, 33 of 64 with carcinoma ex pleomorphic adenoma, 20 of 40 with acinic cell, and 6 of 16 with unspecified carcinomas. Tumor histology, anatomic site, race, and sex were important predictors of survival. A possible relationship between cutaneous neoplasms and salivary carcinoma is discussed.

84–0773. Thorarinsson, H.; Hallgrimsson, J. **Carcinoma of the Lung in Iceland: 1931-1974.** *Environmental Research* 32(2): 414-431, December 1983.

The rising incidence of lung cancer in Iceland is definitely correlated with the increased sales of cigarettes that began some 20 years ago; the vast majority of lung cancer patients with known smoking habits were heavy smokers. In the small cell anaplastic group, there were no nonsmokers. Between 1931 and 1974, 464 cases of primary lung cancer were diagnosed; 34 cases between 1931 and 1954, 156 between 1955 and 1964, and 274 between 1965 and 1974. During these years, the male/female ratio decreased markedly (from 2.8:1 to 1.5:1), due at least in part to the increase in smoking among females. Smoking among females also occurs mainly in urban areas, accounting for higher lung cancer rates among urban women. Between 1955 and 1974, 143 major surgical procedures were performed, 68 exploratory thoracotomies and 75 resections. Of the resections 58.7 percent were pneumonectomies. There were three surgical deaths in the resected series, for a mortality of 4 percent. Pneumonectomy carried a mortality of 6.8 percent, but lobectomies or segmental resections carried no mortality. In the resected series, the absolute 5-year survival rate is 29.3 percent. Patients with epidermoid lesions have the most favorable prognosis. (Auth. Abs. Mod.)

84–0774. Walker, W. J. **Lung Cancer in Women. (Letter).** *New England Journal of Medicine* 310(4): 259-260, January 26, 1984.

While agreeing with an earlier editorial concerning lung cancer in women (*New England Journal of Medicine* 309(7): 428-429, August 18, 1983), the writer is more optimistic that past efforts to reduce cigarette smoking have not been in vain. Because of the change in coding of lung cancer mortality between the seventh and eighth editions of the *International Classification of Diseases*, a comparability ratio of 1.0316 should be applied to mortality rates calculated before 1968. Such figures indicate the death rate for lung cancer in white women peaked around 1970. It is also noted that, while the decrease in smoking rates is not impressive, the tobacco content of cigarettes declined 22.5 percent between 1959 and 1981, which may be responsible for the slower rate of increase in lung cancer mortality in both sexes. Reduction or cessation of smoking could help reverse the trend. See also 84-0119.

84–0775. Winkelstein, W., Jr.; Shillitoe, E. J.; Brand, R.; Johnson, K. K. **Further Comments on Cancer of the Uterine Cervix, Smoking, and Herpesvirus Infection.** *American Journal of Epidemiology* 119(1): 1-8, January 1984.

Data from various studies are presented as evidence that cigarette smoking is an important risk factor for cervical cancer. The biologic plausibility of such an association is supported, while the likelihood that the association is spurious due to an unknown confounding factor is shown to be small, since major recent studies of cervical cancer risk factors have been rigorous and unbiased. Preliminary data on the possibility of an interaction between cigarette smoking and herpes virus infection are included. (Auth. Abs.)

NEOPLASTIC DISEASES

84–0776. World Health Organization. **Le Cancer du Poumon: Situation Actuelle en Matiere de Prevention et de Lutte. [Lung Cancer: Present Status of Prevention and Control.]** *Bulletin de l'Organization Mondiale de la Sante* 61(1): 23-34, 1983, French.

Updated information, from the World Health Organization meeting held in Geneva in November 1981, on the epidemiology, pathologic anatomy, diagnosis, treatment, early detection, and primary prevention of lung cancer, is reviewed. Groups at particularly high risk of lung cancer are smokers; smokers continuously exposed to other carcinogenic agents such as asbestos and radiation; and, apparently, persons exposed to passive smoke. It is stated that elimination of smoking, more than any other measure, would affect the incidence of lung cancer. The initiation of primary prevention programs in different countries, education about smoking of general populations and target groups, the dissemination of information on smoking, antismoking legislation, research on passive smoking, and the development of "light" cigarettes (with low levels of toxic substances) are reviewed. Priority research topics on primary prevention are suggested, noting that smoking control is the most promising approach to primary prevention. A long-term program for smoking control is recommended.

84–0777. Yu, M. C.; Mack, T.; Hanisch, R.; Peters, R. L.; Henderson, B. E.; Pike, M. C. **Hepatitis, Alcohol Consumption, Cigarette Smoking, and Hepatocellular Carcinoma in Los Angeles.** *Cancer Research* 43 (12, Part 1): 6077-6079, December 1983.

Smoking, independently associated with primary hepatocellular carcinoma (PHC), is estimated to be responsible for approximately 20 percent of cases occurring in Los Angeles. Information from interviews with 78 PHC patients (50 males, 28 females) aged 70 years or younger at diagnosis, and 78 age-, sex-, and race-matched neighborhood controls included usual dietary and drinking habits, cigarette smoking habits, prior medical conditions (including a history of hepatitis or prior exposure to blood products), and occupational history. Current smokers of more than one pack/day had 2.6 times the relative risk (RR) of acquiring PHC compared with nonsmokers. Alcohol consumption was also a significant risk factor for PHC; individuals who drank 80 g or more of ethanol/day had a RR of 4.2 compared with those drinking less than 10 g/day. In addition, a history of hepatitis (RR = 13.0) or blood transfusions (RR = 7.0) were significant risk factors for PHC. Each factor remained significant after adjustment was made for the others. (Auth. Abs. Mod.)

NON—NEOPLASTIC RESPIRATORY DISEASES

84–0778. Beck, G. J.; Maunder, L. R.; Schachter, E. N. **Cotton Dust and Smoking Effects on Lung Function in Cotton Textile Workers.** *American Journal of Epidemiology* 119(1): 33-43, January 1984.

Both smoking and exposure to cotton textile dust significantly influenced lung function and were found to be additive and often equally important. Because both smoking and exposure to cotton textile dust have been associated with the development of chronic obstructive lung disease, the relative importance of the two effects was examined, based on a cross-sectional study of white active and retired cotton textile workers 45 years and older seen in 1973 in Columbia, South Carolina. Subjects had worked in the mills an average of 35 years. A questionnaire was completed and an expiratory flow volume curve obtained. Standard pulmonary function parameters including the forced vital capacity (FVC), the forced expired volume in 1 sec (FEV$_1$), and the maximum expiratory flow at 50 percent (MEF$_{50}$), and at 25 percent (MEF$_{25}$) were recorded. White residents 45 years and older from three communities studied from 1972 to 1974 were used as controls. A two-way analysis of variance examined both the effects of cotton dust and smoking on lung function. When one effect was more important than the other, it was in FVC and FEV$_1$ for cotton dust and in MEF$_{50}$ and MEF$_{25}$ for smoking. (Auth. Abs. Mod.)

84–0779. Beck, G. J.; Schachter, E. N.; Maunder, L. R.; Schilling, R.S.F. **A Prospective Study of Chronic Lung Disease in Cotton Textile Workers.** *Annals of Internal Medicine* 97(5): 645-651, November 1982.

Chronic lung disease was found to be independent of smoking habits in an assessment of respiratory symptoms and lung function in both active and retired cotton textile workers and age-matched controls over a 6-year period. In the controls, only 16 percent of the men but 43 percent of the women were smokers, compared with 27 percent of the male and 31 percent of the female cotton workers. Exposure by pack-years, however, was similar in all smoking categories in the two groups. The textile workers had higher prevalence and attack rates of respiratory symptoms compared with controls even when smoking habits were taken into account. Chronic bronchitis developed in 16 percent of all cotton textile workers compared with only 1 percent of controls, and cotton workers suffered a larger loss of lung function over the 6 years of followup. Male workers lost 42 ml/yr of forced expiratory volume in 1 sec, (male controls lost only 25 ml/yr). Similar differences were seen in women, and in both men and women who were nonsmokers. Retired cotton textile workers had more symptoms and disability than active workers. It is concluded that chronic lung disease is not only irreversible, but may progress even after exposure to cotton dust has ended. (Auth. Abs. Mod.)

84–0780. Damodar, A. D.; Sharma, P. N. **Changes in Ventilatory Capacities Due to Exposure to Talc Dust.** *Indian Journal of Chest Diseases and Allied Sciences* 25: 21-24, January-March 1983.

Smoking appears to play a complementary role in lung function decline among industrial talc-exposed workers. In 202 workers and 101 nonexposed controls analyzed for smoking habits, age, dust exposure, and ventilatory capacity, it was found that lung function values declined with age, but the differences between the dust-exposed and nonexposed groups were not significant. Among nonsmokers, lung function did not differ significantly between the experimental and control subjects. However, among light, medium, and heavy smokers, lung function values in dust-exposed workers were significantly lower compared with controls. Indices of lung capacity also declined with duration of dust exposure after the first 5 years of employment.

84–0781. Gupta, V. K.; Pande, J. N.; Guleria, J. S. **Ventilatory Response to Carbon Dioxide in Normal Subjects.** *Indian Journal of Chest Diseases and Allied Sciences* 25: 10-15, January-March 1983.

Ventilatory response to carbon dioxide (CO_2) did not differ significantly between smokers ($n=15$) and nonsmokers among 63 young, healthy Indian subjects (54 males, 9 females). Response to CO_2 was measured by the rebreathing method. Respiratory center sensitivity to CO_2 varied widely and was significantly lower in females compared with males. Also, the threshold of CO_2 stimulus (mean intercept) was lower in females compared with males. The slope of the ventilatory response showed no significant correlation with vital capacity, age, or standing height. When compared to other ethnic groups, respiratory center sensitivity to CO_2 in Indians was found to be higher than that of normal New Guineans, but less than the reported values in Caucasians. These differences, however, were insignificant if the slope was divided by vital capacity. These findings may be relevant to the CO_2 accumulation in patients with chronic obstructive lung disease in India. (Auth. Abs. Mod.)

84–0782. Idell, S.; Cohen, A. B. **Alpha-1-Antitrypsin Deficiency.** *Clinics in Chest Medicine* 4(3): 359-375, September 1983.

Review of the current state of knowledge of the biochemistry of α_1-antitrypsin, its role in defending the lung against proteolytic injury, clinical expressions of deficiency, epidemiology and natural history of deficiency states, and new potential forms of therapy are presented. Findings suggest that cigarette smoking inactivates lung antiproteases, and that oxidation is responsible for inactivation of α_1-antitrypsin with cigarette smoking. Lung washes from smokers show a twofold reduction in the functional inhibitory activity of α_1-antitrypsin compared with lung washes from nonsmokers. Early lesions in smokers consist of collections of macrophages in small airways; these cells secrete hydrogen peroxide, which enhances the oxidative capacity of smoke and its ability to inactivate α_1-antitrypsin. Therefore, the area around the lesions may be particularly lethal to α_1-antitrypsin, possibly explaining development of centrilobular emphysema in smokers. Additional evidence indicates that smoking may reduce the resynthesis of damaged elastin. Formation of desmosine, a major cross-link in elastin, is inhibited in a cell-free system exposed to aqueous dilutions of cigarette smoke. It is indicated that exposure to tobacco smoke retards elastin repair in vivo by more than 40 percent. Proposed biochemical links between cigarette smoking and emphysema pathogenesis are illustrated.

84–0783. Keller, R. **Chronische Lungenkrankheiten Beim Raucher. [Chronic Lung Diseases in Smokers.]** *Therapeutische Umschau* 40(2): 133-138, February 1983, German.

Prolonged duration of smoking is the most important risk factor in the development of chronic lung diseases such as bronchitis and emphysema. Mortality and morbidity among smokers are six to eight times that of nonsmokers. Toxic substances in the gaseous phase of cigarette smoke produce inflammation of the small airways (small airways disease), disturbed mucocele clearance, reduced resistance to infection, and bronchial stenosis thus paving the way for emphysema of the lungs, which can be diagnosed in early stages. Subsequently, inflammatory and degenerative damage results, with mucoglandular hyperplasia, destruction, and atrophy of the mucous membrane. The extent and prevalence of chronic lung disease depends on the duration and intensity of the smoking habit as well as additional damaging environmental influences and endogenous factors, but are largely independent of sex or the cigarette tar and nicotine content. Stopping smoking at any stage results in subsiding of bronchial symptoms, lessening of lung function disturbance, and improved prognosis. From a preventive point of view, early diagnosis of chronic bronchitis and broad-based motivation to stop smoking are to be supported.

84–0784. Kowalski, J.; Spychala, A. **Sredni Czas Przemieszczania (*Mean Transit Time* — MMT) — Nowy Test Nasilonego Wydechu. [Mean Transit Time (MTT) — New Test of Forced Expiration.]** *Pneumonologia Polska* 51(1): 1-11, 1983, Polish.

Mean transit time (MTT) and forced expiratory volume in 1 sec percent of forced vital capacity ($FEV_1/FVC \times 100$) were evaluated in 100 healthy subjects (all males, 45 nonsmokers and 55 chronic smokers). FEV_1 was measured using a conventional spirograph. In smokers, MTT was higher (0.70 ± 0.14, $FEV_1/FVC \times 100$ 82.9 ± 4.7). The differences between mean values were statistically significant. A high linear correlation was found between MTT and $FEV_1/FVC \times 100$ for smokers ($r=0.909$) and nonsmokers ($r=0.868$). Using student's "t" test, significant differences were found between angles of linear correlation of MTT and $FEV_1/FVC \times 100$ for both groups. In 32 smokers (58.2 percent) and 24 nonsmokers (53 percent), a 6-second recording of forced expiration was enough for approximate estimations of MMT, FVC, and $FEV_1/FVC \times 100$ values. (Auth. Abs. Mod.)

84–0785. Kulpati, D.D.S.; Sharma, R. K.; Chauhan, M. R. **A Study of Small Airways Function by Closing Volume and Density Dependent Flow Volume Curves.** *Indian Journal of Chest Diseases and Allied Sciences* 25: 4-9, January-March 1983.

Symptomatic smokers among 100 male subjects (aged 20 to 50) had more peripheral airways dysfunction compared with asymptomatic smokers and asymptomatic asthmatics. Small airways functions were studied in groups with nearly normal forced expiratory volume in 1 sec, consisting of 25 normal healthy nonsmokers (group A), 25 asymptomatic smokers (group B), 25 asymptomatic asthmatics (group C), and 25 symptomatic smokers (group D). Compared with group A, group C patients had significantly higher closing capacity/total lung capacity, whereas both closing volume/vital capacity and closing capacity/total lung capacity were increased in group D. Asymptomatic smokers had a shorter history of smoking (2 to 3 pack-years), which may explain why none had abnormal closing volume/vital capacity (in contrast with previous studies). Maximum midexpiratory flow rates and forced expiratory flow were significantly low in groups C and D only. However, density-dependent flow volume curves with helium-oxygen mixture ($He-O_2$) separated all groups from healthy adults and was found to be the most sensitive parameter of peripheral airway dysfunction, being 56, 64, and 76 percent in groups B, C, and D, respectively.

84–0786. Main, D. M.; Hogan, T. J. **Health Effects of Low-Level Exposure to Formaldehyde.** *Journal of Occupational Medicine* 25(12): 896-900, December 1983.

Data for 21 subjects exposed to formaldehyde at levels between 0.12 and 1.6 ppm showed no significant differences in forced expiratory volume in 1 sec or forced vital capacity when compared with 18 workers not exposed to formaldehyde, regardless of smoking status. There were 17 smokers and 4 nonsmokers in the exposed group, and a similar percentage among the nonexposed control group. Eight of the nonexposed and six of the exposed group reported working in an area filled with cigarette smoke during most of the day. Symptoms of eye and throat irritation, headache, fatigue, irritation of the nose, chest tightness, and shortness of breath were more common among formaldehyde-exposed workers, although spirometry revealed no decrease in ventilatory function. Results indicate that adverse health effects occur from exposure to formaldehyde at levels between 0.12 and 1.6 ppm.

84–0787. Morgan, W.K.C.; Clague, H. W.; Vinitski, S. **On Paradigms, Paradoxes, and Particles.** *Lung* 161(4): 195-206, June 1983.

Recent findings confirm apical ventilation defect in cigarette smokers, representing subclinical emphysema or early small airways disease. Centrilobular emphysema, commonly associated with smoking, has an apical predominance, possibly because inhaled irritants predominate in areas with a high ventilation/perfusion ratio, i.e., the lung apex. Emphysema associated with α_1-antitrypsin deficiency occurs in areas of highest perfusion, i.e., the lung base. Other possible factors in the apical localization of centrilobular emphysema are discussed. High flow rates at the start of inspiration are likely to encourage central airway deposition by impaction and may explain why bronchial carcinoma is most common at the bifurcation sites in the upper right lobe. It is suggested that cigarette smoke is carried predominantly to the upper lobes, where most of it remains until the end of the subsequent exhalation, resulting in a longer residence time in the upper zones. Following exhalation, the upper lobe alveoli have a greater volume and a greater amount of smoke probably persists, leading to more mixing of smoke with alveolar air due to diffusion. Retention of cigarette smoke may also be exaggerated by the small airways disease that exists in the upper zones. Additional aspects of particle deposition and clearance from lungs are reviewed regarding their role in lung disease etiology and anatomical localization of the earliest pathological lesion.

84–0788. Nancy, N. R.; Rai, U. C. **A Study of Forced Expiratory Spirogram in South Indian Beedi Smokers and Cigarette Smokers.** *Indian Journal of Chest Diseases and Allied Sciences* 25: 25-30, January-March 1983.

Pulmonary spirographic measurements of 80 subjects (25 nonsmokers, 25 bidi smokers, and 30 cigarette smokers) indicated that peak expiratory flow and forced expiratory flow values (measured at 3 lung volumes, 70 to 80 percent, 45 to 55 percent, and 25 to 75 percent) were significantly lower among smokers compared with controls. Among smokers, cigarette users had lower flow rates than bidi smokers. Forced expiratory volume in 1 sec, expressed as a percentage of forced vital capacity, was significantly impaired only in cigarette smokers. The flow rates at different lung volumes, considered to be a more sensitive index of airways obstruction, indicate that a functional impairment is present in bidi smokers as well, although less pronounced than in cigarette smokers. (Auth. Abs. Mod.)

84–0789. Okeson, G. C. **Pulmonary Dysfunction and Surgical Risk. How to Assess and Minimize the Hazards.** *Postgraduate Medicine* 74(5): 75-77, 80, 82-83, November 1983.

Incidence of postoperative pulmonary complications increases directly with daily cigarette consumption above a threshold of 8 to 10 pack-years. Postoperative deterioration of lung function is considerable, resulting in hypoxia and atelectasis. After abdominal surgery, a patient's vital capacity may decrease 25 to 50 percent, with the maximal decrease during the first 2 postoperative days. Chronic smoking produces a variety of pathophysiologic changes in the lung, all of which also predispose to postoperative atelectasis and pneumonitis. If the patient is to have thoracic or upper abdominal surgery and is a smoker, or obese, or over age 70, a preoperative evaluation of lung function is recommended. The high risk of such pulmonary complications can be reduced with a preoperative therapeutic program that includes cessation of smoking, weight loss, and other changes.

84–0790. Ploysongsang, Y. **The Lung Sounds Phase Angle Test for Detection of Small Airway Disease.** *Respiration Physiology* 53(2): 203-214, August 1983.

The sequence of breathing sounds in various parts of the lung during inspiration (lung sounds ventilation sequence index θ) was measured in 15 smokers and 19 nonsmokers. Various other lung function parameters, and pulmonary resistances and compliances at differing breathing frequencies were also determined. The mean ventilation sequence, or phase angle, θ was 3.3° for nonsmokers compared with 8.1° in smokers. Among the smokers, 73 percent had abnormal phase angles ($\theta > 5°$), suggesting that smokers' lung units are inspiring more out of phase than the nonsmokers'. Also, the ratio of dynamic to static compliance was lower in smokers than in nonsmokers. Both the ratio of forced expiratory volume to forced vital capacity, and forced expiratory flow, were significantly lower in the smokers, but were within the normal range. The other lung function indices did not vary significantly between the two groups. It is concluded that θ is an index of the inhomogeneity of lung time constants, and is an appropriate noninvasive test for detecting small airways disease.

84–0791. Pratt, P. C.; Lynn, W. S. **Lung Disease in Cotton Workers. (Letter).** *Annals of Internal Medicine* 99(2): 278, August 1983.

Further comments are supplied in reply to the original authors' rebuttal to criticism of an earlier report (*Annals of Internal Medicine* 98(4): 554-555, April 1983), noting that data do not support conclusions of a continued deterioration in lung function among retired cotton workers, regardless of smoking status. It is suggested that smokers alone appear to be responsible for the decline in lung function values. Originally, the retirees were compared with controls, divided according to smoking status. However, when followup data on lung function of retirees were compared with values from 6 years earlier, the mean forced expiratory volume in 1 sec (FEV_1) in ex- and nonsmokers, originally 88.9 percent of predicted, was 90.0 percent. Smokers, in contrast, who originally had a mean FEV_1 of 79.3 percent, had only an average 69.2 percent at followup. Thus, the earlier suggestion that nonsmoking cotton workers' impairment may regress after retirement is given some support. See also 84-0792.

84–0792. Pratt, P. C.; Lynn, W. S.; Beck, G. J.; Schachter, E. N.; Maunder, L. R.; Schilling, R.S.F.; Battigelli, M. C. **Lung Disease in Cotton Textile Workers. (Letters).** *Annals of Internal Medicine* 98(4): 554-555, April 1983.

A previous article (*Annals of Internal Medicine* 97(5): 645-651, November 1982) is criticized for implying that progression of chronic lung disease in nonsmoking cotton textile workers occurs after retirement. The data in the report were not separated according to smoking status, and it was also noted that age alone was not a reason for retirement. Thus, the retirees evaluated were a self-selected group, possibly with greater impairment and having a sizable proportion of smokers. The authors reply that smoking rates were 25 percent among the male and 17 percent in the female retirees, a lower proportion than in the active workers. Also, regression analysis showed greater lung function losses in the retired workers even after adjustment for smoking, implying a continued deterioration in the absence of cotton dust exposure. It is stated that the evidence is consistent with irreversible chronic lung disease in cotton textile workers. See also 84-0779.

84–0793. Rosenthal, A.; Solomons, N. W. **Time-Course of Cigarette Smoke Contamination of Clinical Hydrogen Breath-Analysis Tests.** *Clinical Chemistry* 29(11): 1980-1981, 1983.

The time-course of the contamination of exogenous hydrogen from cigarette smoke on postprandial breath hydrogen concentration was evaluated in 10 healthy subjects, 6 regular smokers (at least 20 cigarettes/day) and 4 occasional smokers. Breath hydrogen values were determined by gas chromatography (1) 10 min, 5 min, and immediately prior to smoking a filter cigarette; (2) during smoking from a sample of exhaled air containing smoke; and (3) 5, 10, and 15 min after extinguishing the cigarette. A 3- to 137-fold increase above basal hydrogen concentrations was produced by exhaled cigarette smoke, but most subjects had re-equilibrated to baseline values within 10 to 15 min after smoking the cigarette. If subjects undergoing clinical hydrogen breath tests cannot refrain from smoking during the duration of the test, an interval of at least 15 min from the end of smoking to the collection of a breath sample should be allowed. (Auth. Abs. Mod.)

84–0794. Schenker, M. B.; Samet, J. M.; Speizer, F. E. **Risk Factors for Childhood Respiratory Disease. The Effect of Host Factors and Home Environmental Exposures.** *American Review of Respiratory Disease* 128(6): 1038-1043, December 1983.

Chest illness before 2 years of age and chest illness in the past year showed a significant positive linear trend with the number of parental smokers (confirmed by logistic analysis). Standardized respiratory disease questionnaires were completed by the parents of 4,071 children 5 to 14 years of age, and risk factors for respiratory symptoms were evaluated. Younger age, male sex, and lower socioeconomic status (SES) were independent risk factors for most respiratory symptoms and illnesses. All respiratory outcomes were significantly more prevalent in children with physician-diagnosed asthma, which was more prevalent in male (4.5 percent) than in female (2.6 percent) children. The greater prevalence of most respiratory symptoms in males did not persist in children with the same asthma status. Children whose questionnaires were completed by fathers were reported to have significantly fewer respiratory symptoms than children with mother-completed questionnaires, suggesting that fathers underreport symptoms for their children. A positive parental history of allergy or respiratory illness was an independent predictor of respiratory symptoms and illnesses in the children. The use of gas cooking stoves, inversely associated with SES, was not an independent risk factor for any respiratory symptoms or illnesses. (Auth. Abs. Mod.)

84-0795. Schenker, M. B.; Speizer, F. E.; Samet, J. M.; Gruhl, J.; Batterman, S. **Health Effects of Air Pollution Due to Coal Combustion in the Chestnut Ridge Region of Pennsylvania: Results of Cross-Sectional Analysis in Adults.** *Archives of Environmental Health* 38(6): 325-330, November-December 1983.

Cigarette smoking was the major determinant of respiratory symptoms in smokers (25 percent of sample), and no independent association with air pollution due to coal combustion was found. Questionnaires were administered to 5,557 adult women in a rural area of western Pennsylvania to evaluate the health effects of air pollution resulting from coal combustion. Air pollution data were derived from 17 air quality monitor sites and stratified to define low, medium, and high pollution areas. Risks of respiratory symptoms were evaluated in a multiple logistic model that adjusted for several potential confounding factors. The risk of "wheeze most days or nights" in nonsmokers residing in the high and medium pollution areas was 1.58 and 1.26 ($p=0.02$), respectively, compared with residents in the low pollution area. An increased risk of grade 3 dyspnea in nonsmokers was associated with sulfur dioxide, but did not achieve statistical significance, and there was no association of cough or phlegm and air pollution in nonsmokers. Data suggest that wheezing may be associated with ambient exposure to sulfur dioxide in nonsmokers, but no effect of sulfur dioxide on cigarette smokers was observed. Chronic cough and phlegm production were not associated with sulfur dioxide at the observed concentrations. (Auth. Abs. Mod.)

84-0796. Sepulveda, M.-J.; Merchant, J. A. **Roentgenographic Evidence of Asbestos Exposure in a Select Population of Railroad Workers.** *American Journal of Industrial Medicine* 4(5): 631-639, 1983.

Radiological abnormalities among 266 current and former railroad workers were related to latency period, age, and occupation, but not to smoking habits. Of the total, 75 percent were over age 60, and 80 percent had fewer than 10 years of railroad-related asbestos exposure. Roentgenographic evidence of asbestosis was found in only six workers (2 percent), whereas 20 percent had one or more pleural changes. Of the 266 participants, 22 percent were current smokers, 44 percent ex-smokers, and 34 percent never smokers. Adjusting for differences between smoking categories for age and years since onset of employment revealed no difference in the prevalence rate of radiological findings among the various smoking categories. While selection factors qualify the results of this study, the findings support the exposure and suggest a past and future history of asbestos mortality and morbidity among steam era railway workers. (Auth. Abs. Mod.)

84-0797. Simard, A.; Vauclair, R.; Cole, P.; Perret, C.; Nault, M.; Paquette, G. **Pulmonary Cytology in Foundry Workers.** *Journal of Chronic Diseases* 36(9): 617-623, 1983.

Although a higher risk of lung cancer among foundry workers was expected, an increase in atypical metaplasia was found only in men over 50 years of age, and followed a linear trend with exposure to smoking. The prevalence of cellular atypias, determined by sputum cytology in 677 foundry workers in Montreal, was 5.3 percent mild, and 0.6 percent moderate atypical metaplasia. The prevalence increased with age and in relation to smoking status: 4.2 percent for nonsmokers to 7.8 percent in those smoking >21 cigarettes/day. Variations in prevalence associated with employment in different parts of the foundry were not statistically significant. When relative risk is calculated by smoking status, however, there is a significant linear trend (1.0 for nonsmokers, 1.5 for ex-smokers, and 1.9 for current smokers).

84-0798. Soutar, C. A.; Gauld, S. **Clinical Studies of Workers Exposed to Polyvinylchloride Dust.** *Thorax* 38(11): 834-839, November 1983.

Data on the clinical and physiological features associated with exposure to polyvinyl chloride (PVC) showed that, among 28 exposed workers with small rounded opacities, complaints of persistent bronchial mucous hypersecretion were more common than among 29 men with similar smoking habits, age, and dust exposures. A study of 18 smokers with the highest PVC dust exposures and lowest adjusted forced expiratory volume in 1 sec (FEV_1) found that some had an observed FEV_1 well below 80 percent of predicted values for nonsmokers, but, with one exception, no clinical or functional differences were found when compared with 18 smokers with low dust exposure and similarly low FEV_1. Among 13 nonsmokers with the highest PVC dust exposure and lowest FEV_1 (adjusted for age, height, weight, and dust exposure), none had an observed FEV_1 less than 80 percent of the predicted value for age and height, and the men did not differ in symptoms, signs, or lung function from nonsmokers with low dust exposure and similarly low FEV_1. The average lung function of men with opacities was only slightly lower than that of the comparative group; the difference between the groups was not significant. It is concluded that the findings are consistent with the suggestion that the radiographic abnormalities caused by PVC dust exposure are not associated with important functional effects or clinical illness. (Auth. Abs. Mod.)

84–0799. Tobin, M. J.; Jenouri, G. A.; Watson, H.; Sackner, M. A. **Noninvasive Measurement of Pleural Pressure by Surface Inductive Plethysmography.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 55(1): 267-275, July 1983.

Use of a noninvasive plethysmographic technique revealed frequency-dependent lung compliance among smokers with small airways disease. Recession of the suprasternal fossa that takes place during inspiration can be monitored with surface inductive plethysmography (SIP). The transducer consists of a flat loop of Teflon-insulated wire coupled to the skin. Excitation by an oscillator circuit leads to change in self-inductance of the wire that is proportional to the cross-sectional area of an imaginary plane enclosed by the loop. Data from both models and humans indicate that movement of the suprasternal fossa is proportional to intrapleural pressure. Static lung compliance during inflation was obtained using a "hull procedure" in six normal subjects and eight patients with lung disease, and both SIP and esophageal pressure gave similar values. Dynamic lung compliance was calculated at respiratory rates from 20 to 60 breaths/min; simultaneous SIP and esophageal balloon values in seven normal subjects and seven patients with lung disease showed close correlation. Frequency dependence of compliance was demonstrated in a group of smokers with small airways disease whereas compliance was not frequency dependent in normals. (Auth. Abs. Mod.)

84–0800. Tollerud, D. J.; Weiss, S. T.; Elting, E.; Speizer, F. E.; Ferris, B. **The Health Effects of Automobile Exhaust. VI. Relationship of Respiratory Symptoms and Pulmonary Function in Tunnel and Turnpike Workers.** *Archives of Environmental Health* 38(6): 334-340, November-December 1983.

Smoking was strongly related to respiratory symptoms of cough and phlegm production among 175 tunnel and turnpike workers (109 smokers, 41 ex-smokers, 24 never smokers) examined for the effect of automobile exhaust on respiratory symptoms and pulmonary function. On two occasions, 3 years apart, a standard respiratory symptom and illness questionnaire was administered, spirometry was performed, and proximal hair lead and blood lead content were measured as biologic indices of automobile exhaust exposure. Smoking was not related to wheezing, breathlessness, bronchial asthma, or frequency of chest colds. When workers were stratified by smoking status, no effect could be seen between high automobile exhaust exposure as measured by a variety of parameters and all of the above respiratory symptoms and illnesses. The level of pulmonary function (forced expiratory volume in 1 sec (FEV_1) and forced vital capacity (FVC)) was not related to past or current exhaust exposure in a cross-sectional analysis when controlled for age, height, and cigarette consumption. In a prospective analysis of 84 workers, the observed changes in FEV_1 and FVC over 3 years were unrelated to exhaust exposure after controlling for age, height, cigarette consumption, and initial level of pulmonary function. (Auth. Abs. Mod.)

84–0801. Tonnel, A. B.; Gosset, P.; Joseph, M.; Fournier, E.; Steenhouwer, F.; Mallart-Voisin, A. **La Reponse Cellulaire Locale aux Agressions du Poumon Profond. [Local Cellular Response to Intra-pulmonary Disease.]** *Revue Francaise des Maladies Respiratoires* 11: 301-315, 1983, French.

Alveolar cell responses to mineral particles, toxic gases, pathogens, antigens, and pollutants such as tobacco smoke are reviewed. The major long-term effects of smoking include (1) an increase in the macrophage-lymphocyte ratio from 85:15 in nonsmokers to 95:5 in smokers; (2) an increase in the number and size of lysosomes; (3) an alteration in the capacity of macrophages to detoxify polycyclic hydrocarbons, with the resulting increase in aryl hydrocarbon hydroxylase; (4) an increase in the migration rate of alveolar macrophages; (5) the alteration of cellular metabolism from increased production of oxidizing agents and metabolism of arachidonic acid; and (6) an increase in T-cells. Overall, tobacco smoke leads to a number of changes in the distribution, function, and morphology of alveolar cells consistent with pulmonary disease.

84–0802. Tos, M.; Moller, K. **Goblet-Cell Density in Human Bronchus in Chronic Bronchitis.** *Archives of Otolaryngology* 109(10): 673-676, October 1983.

Goblet cell density was found to be significantly higher in smokers than in nonsmokers; of the six patients who did not smoke, five were in the normal group, and only one had mild bronchitis. Among the 44 smokers, 11 were in the normal group and 17 had mild bronchitis. All 16 patients with chronic bronchitis were smokers. In the 50 patients studied, up to six mucosal biopsy specimens were taken during bronchoscopy from the medial part of the trachea and from the main and lobe bronchi, and the density of goblet cells was studied using the whole-mount method. No significant differences in density were found between the trachea and the main or lobe bronchi in the normal patients or in the cases with abnormalities. In the normal patients, the interindividual median density was 127 cells/field corresponding to 7,200 cells/sq mm; in chronic bronchitis, there were 174 cells/field, corresponding to 9,800 cells/sq mm. (Auth. Abs. Mod.)

84–0803. Wright, J. L.; Churg, A. **Morphology of Small-Airway Lesions in Patients With Asbestos Exposure.** *Human Pathology* 15(1): 68-74, January 1984.

Small airways lesions in asbestos-exposed smokers were found to be distinguishable from the lesions of nonasbestos-exposed smokers. The morphologic changes in the small airways of 15 patients (13 smokers) who had been exposed to asbestos and had abnormalities of the respiratory bronchioles and alveolar ducts were examined and compared with changes in the small airways of a matched group of cigarette smokers. Lesions were evaluated using a visual grading scheme to determine pathologic severity of fibrosis, inflammation, and pigmentation in these airways. The membranous bronchioles of both asbestos-exposed and cigarette-exposed patients showed the deposition of fibrous tissue and pigment. The lesions were morphologically identical, although more severe in the asbestos group (67 percent of airways) than in the smoking group (19 percent of airways). A distinctive lesion, consisting of fibrosis often accompanied by pigment, was found in 74 percent of the respiratory bronchioles in asbestos-exposed patients, compared with 6 percent of respiratory bronchioles in smokers. Similar changes were found in 49 percent of alveolar ducts in the asbestos group and 1 percent of the ducts in the smoking group. Asbestos bodies were found in only 11 of 15 cases. (Auth. Abs. Mod.)

See also, **84–0712, 84–0847, 84–0883**

CARDIOVASCULAR DISEASES

84–0804. Armstrong, B. K.; Margetts, B. M.; Masarei, J.R.L.; Hopkins, S. M. **Coronary Risk Factors in Italian Migrants to Australia.** *American Journal of Epidemiology* 118(5): 651-658, November 1983.

The small differences in coronary risk factors between 177 Italian immigrants and 176 age-matched native-born Australians appear insufficient to explain differences found in ischemic heart disease mortality. There were slightly more current smokers among Italian men (53.6 percent) compared with Australian men (47.0 percent), while the opposite was true for women; 18.4 percent of Italians and 31.0 percent of the Australians were current smokers. However, the mortality from ischemic heart disease in Italians in Italy and in Italian immigrants to Australia is between one-third and two-thirds that found in native-born Australians. Other risk factors measured were height, weight, blood pressure, serum total cholesterol, high density lipoprotein (HDL) cholesterol, serum apo-HDL, physical activity, and drinking habits. The Italian men had a lower mean systolic and diastolic blood pressure than the Australian men, but Italians of both sexes had higher mean body mass indices than Australians. Italian women had a significantly lower mean serum HDL cholesterol concentration than Australian women. The blood pressure differences between Italians and Australians were increased by adjustment of the means for differences between the two groups in age and obesity. A lower salt intake in Italians is one possible explanation for the comparatively low blood pressures, and may, perhaps, help explain the difference in ischemic heart disease mortality. (Auth. Abs. Mod.)

84–0805. Burch, P.R.J.; Daly, L. E.; Mulcahy, R.; Graham, I. M.; Hickey, N. **Effect of Stopping Smoking After Unstable Angina and Myocardial Infarction. (Letters).** *British Medical Journal* 288(6411): 146, January 14, 1984.

The conclusion of a recent study (*British Medical Journal* 287(6388): 324-326, July 30, 1983) that "stopping smoking is the most effective single action in the management of patients with coronary heart disease" is questioned on methodological grounds. Genetic predisposition to coronary heart disease, an important risk factor, can be controlled by randomization of a large study group into intervention and normal care subgroups. A 10-year followup of such a trial revealed no significant difference in mortality. Also, analysis of temporal smoking trends and fatal ischemic heart disease revealed no appreciable causal component. The authors reply that ethical considerations make randomized trials unacceptable practice, but that their study revealed no confounders to contradict the conclusion that those who continued to smoke after myocardial infarction were 2.4 times as likely to die as those who quit. They state that the two studies cited referred to primary, not secondary, prevention, and as such are irrelevant. See also 84-0171.

84–0806. Campbell, W. B.; Slack, R.W.T.; Clifford, P. C.; Smith, P.J.B.; Baird, R. N. **Vasectomy and Atherosclerosis: An Association in Man?** *British Journal of Urology* 55(4): 430-433, August 1983.

While statistical analysis of distensibility at the posterior tibial arteries of 49 vasectomized men and 33 controls showed reduced distensibility in vasectomy patients, significantly higher ω_0 values (an index of stiffness of the lower limb arterial segment) were found in nonsmokers compared with smokers. The distensibility of the lower limb arteries was investigated by an ultrasonic technique. Acceleration in the development of atherosclerosis in animals following vasectomy has been attributed to an immunological mechanism. Immunological changes are also common in men following this operation, but epidemiological surveys have failed to reveal evidence of an increase in arterial disease. Distensibility at the posterior tibial arteries was studied by Laplace transform analysis of Doppler waveforms. Results lend support to the possibility that vasectomy may accelerate the development of peripheral arterial disease in man. (Auth. Abs. Mod.)

84–0807. Carlsson, I.; Wennmalm, A. **Effect of Cigarette Smoking on Reactive Hyperaemia in the Human Finger.** *Clinical Physiology* 3(5): 453-460, October 1983.

Basal finger blood flow and reactive hyperemia were significantly reduced (by 35 and 55 percent, respectively) following cigarette smoking. The effect of cigarette smoking on reactive hyperemia that develops following release of arterial occlusion in human skin was investigated in five men and seven women (20 to 45 years of age) and compared with effects of oral administration of indomethacin or nicotine, or of smoking nicotine-free cigarettes. Finger blood flow was determined, using venous occlusion plethysmography, in the basal state and after 5 minutes of arterial occlusion. All subjects were studied before and after smoking two tobacco cigarettes, two herbal (nicotine-free) cigarettes, or after chewing a nicotine-containing gum. The determinations before and after tobacco smoking were repeated after administration of indomethacin. In separate series, the effects of smoking on heart rate and systemic blood pressure were recorded. The reactive hyperemia after administration of indomethacin in combination with cigarette smoking did not differ from that obtained after cigarette smoking alone. The reactive hyperemia was not affected by oral administration of nicotine, or by smoking two herbal cigarettes. Cigarette smoking elicited increases in heart rate and systemic blood pressure that were of similar magnitude before and after indomethacin. It is concluded that cigarette smoking elicits an inhibitory effect on the reactive hyperemia in the human finger, probably not caused by nicotine, and seems to act via blockade of the vascular relaxation normally mediated by locally formed cyclo-oxygenase products. (Auth. Abs. Mod.)

84–0808. Cupples, L. A.; Heeren, T.; Schatzkin, A.; Colton, T. **Multiple Testing of Hypotheses in Comparing Two Groups.** *Annals of Internal Medicine* 100(1): 122-129, January 1984.

The number of cigarettes smoked/day was one of 10 parameters analyzed for 2,248 men in the Framingham Heart Study (640 of whom developed coronary heart disease), to compare results of multiple tests of hypotheses on individual variables with multivariate analysis. From the mean and standard deviation of each variable and the p values, all parameters except hemoglobin, and perhaps amount smoked ($p < 0.06$) were long-range predictors of coronary heart disease. Results were compared with three procedures for the two groups: Hotelling's T^2, discriminant analysis, and logistic regression. Using the logistic regression model, the relative risk for those who smoked two packs/day was increased 1.6 times compared with not smoking, when all other characteristics were equal. Persons who smoked two packs/day and whose blood pressure was 170 mm Hg had a risk 2.9 times that of nonsmokers whose blood pressure was 110, when all other characteristics were equal. Discriminate analysis yielded almost identical results. The underlying assumptions and relative merits and disadvantages of the three multivariate methods are reviewed, and recommendations made about which method to use in various circumstances.

84–0809. Daugherty, S. A. **Description of the Enumerated and Screened Population.** *Hypertension* 5(6, Part 2): IV-1—IV-43, November-December 1983.

Information on smoking habits is included among the characteristics of 10,500 subjects who participated in the Hypertension Detection and Follow-Up Program (HDFP) that are presented in tables, organized by sex, race, and age. The HDFP is a 5-year, national, multicenter, randomized clinical trial of the effectiveness of antihypertensive therapy in reducing mortality from all causes among hypertensives. Two tables charting the mean blood pressures (systolic and diastolic) of the smoking subjects are included. Approximately 37 percent of the screened population smoked cigarettes. The rate was higher among those not treated for hypertension at baseline than among those treated. The smoking rate increased with increasing diastolic blood pressure level, decreased with age, and was higher in blacks compared with whites, and in men compared with women. (Auth. Abs. Mod.)

84–0810. Downey, H. F.; Crystal, G. J.; Bashour, F. A. **Regional Myocardial Blood Flow During Nicotine Infusion After Chronic Coronary Artery Occlusion: Effect of β-Adrenergic Blockade.** *Journal of Cardiovascular Pharmacology* 5(4): 685-690, 1983.

Data indicate that nicotine-induced increase in blood flow is blunted in a collateral-dependent (CD) region and that, during nicotine infusion, β-adrenergic blockade unmasks coronary vasoconstrictor mechanisms preventing increased flow to either normal or CD regions, despite increased perfusion pressure and augmented myocardial oxygen demands. The left ventricular free wall (LVFW) was rendered CD in eight dogs by gradual occlusion of the left anterior descending coronary artery. Six to 8 weeks later, the dogs were anesthetized and regional myocardial blood flow was measured during (1) control conditions, (2) nicotine alone, and (3) nicotine after β-adrenergic blockade with propranolol. During control conditions, mean transmural flow was similar in CD, border, and normal regions of the LVFW. Nicotine alone increased flow in all regions of the LVFW, with normal (+104 percent) greater than CD (+56 percent). These changes in flow were accompanied by increases in mean arterial pressure (+34 percent) and mean aortic flow (+54 percent). Nicotine after β-adrenergic blockade appreciably raised mean arterial pressure (+83 percent) and mean left atrial pressure (+307 percent), but caused no increase in flow to any region of the LVFW. (Auth. Abs. Mod.)

84–0811. Escudero de la Pena, J. **Tabaquismo y Enfermedades Cardiovasculares. [Smoking and Cardiovascular Disease.]** *Revista Medica* 21(2): 135-137, March-April 1983, Spanish.

The first proof of an association between smoking and cardiovascular death came from retrospective studies of bronchogenic carcinoma, and was confirmed by results of the Framingham study. It is now universally accepted that smoking, arterial hypertension, and hypercholesterolemia are the three most important factors in coronary disease. Two mechanisms are involved: coronary changes (spasm, sclerosis, and thrombosis) and myocardial dysfunction, produced by the combined action of nicotine and carbon monoxide. Smoking is a health-damaging habit, producing respiratory, digestive, hematological, urinary, gynecological, and especially cardiovascular changes. In the United States, smoking is responsible for 11 million chronically ill persons, 77 million lost workdays, and 306 million reduced-capacity days each year. Smoking accelerates the degenerative process of the arteries; consequently, smokers acquire cardiovascular complications at an early age. It is not unreasonable to say that "every minute one smokes is one minute less of life."

84–0812. Filikowski, J.; Grzegorzewski, M. **Epidemiological and Social Aspects of Heart Infarct in Seamen.** *Bulletin of the Institute of Maritime and Tropical Medicine in Gdynia* 33(1/2): 5-12, 1982.

Of 129 sailors who suffered a myocardial infarction (MI), 106 (82.2 percent) were smokers compared with only 66.6 percent of seamen as a group. Cardiovascular risk factor information was gathered from Polish Merchant Marine seamen who had suffered MIs. Most of the infarctions had occurred in subjects aged 41 to 60, after a period of work at sea of 16 to 25 years. Traditional risk factors were present in all but four patients: smoking and hypertension in 6 subjects; smoking and obesity in 12; smoking, obesity, and hypertension in 36; and 4 with hypertension, diabetes, obesity, and smoking. Chronic alcoholism was also diagnosed in 11; 46 were heavy drinkers. Most of the seaman suffered MI while on land (48.8 percent), usually at home on leave, while only 20.15 percent suffered an MI aboard the ship at sea. It is suggested that the high proportion of MIs suffered on land were due partly to conditions to which the seamen were less adapted after so many years at sea. It might also help explain the high percentage of seamen who returned to work at sea following MI (88.37 percent), which is much higher than land employees who return to work after MI (between 30 and 69 percent).

84–0813. Garg, J. P.; Gupta, R. S.; Agrawal, M. P.; Bhandaril, V. M. **Effect of Smoking on Serum Lipids and Lipoproteins in Healthy Subjects, and Patients of Old Myocardial Infarction and Hypertension.** *Indian Journal of Medical Sciences* 37(4): 63-67, April 1983.

Data suggest that the manner in which chronic smoking alters the serum lipid and lipoprotein profile of healthy smokers makes them susceptible to accelerated atherosclerosis. However, once clinical ischemic heart disease or hypertension develops, smoking ceases to have a significant effect, suggesting that the best time to quit smoking is before the development of clinical cardiovascular disease. Results from 150 males (50 healthy subjects, 50 myocardial infarction (MI) patients, 50 hypertensives) found that all MI and hypertension patients (25 smokers, 25 nonsmokers in each group) had significantly higher serum cholesterol and triglycerides compared with healthy subjects. Differences between smoker and nonsmoker MI and hypertension patients were not significant. It is possible that in such patients, other factors affecting serum lipids and lipoproteins are so pronounced that the contribution of smoking becomes insignificant.

84–0814. German, A. K.; Belinskiy, V. P. **Vliyaniye Kureniya na Pokazateli Gemodinamiki i Kislorodnogo Balansa Krovi u Zdorovykh. [The Effect of Smoking on the Hemodynamic Indicators and the Oxygen Balance of the Blood in Healthy Persons.]** *Vrachebnoye Delo* 2: 78-82, 1983, Russian.

Oxygen balance and other hemodynamic parameters were studied in 60 healthy smokers, aged 23 to 32 years. Results indicate that the nicotine and carbon monoxide in tobacco smoke has an active effect on the cardiovascular system. Long-term smoking effects were characterized by a tendency for the blood volume to rise as a result of an increased impact volume, and a concomitant increase in diastolic pressure in the major circulation and systolic pressure in the minor circulation. Consequently, long-term smoking reduces the efficiency of oxygen utilization by the myocardium and other tissues. This discrepancy between increased demand and reduced efficiency of oxygen utilization in smokers may be an early factor in developing arteriosclerosis.

84–0815. Green, D.; Stone, N. J.; Krumlovsky, F. A. **Putative Atherogenic Factors in Patients With Chronic Renal Failure.** *Progress in Cardiovascular Diseases* 26(2): 133-144, September-October 1983.

Cigarette consumption is included in a review of the multiple, often interacting factors that contribute to the high incidence of atherogenesis in uremic patients. Smoking is a major risk factor in patients undergoing hemodialysis. Endothelial damage occurs from the cytotoxic effects of aromatic hydrocarbons, elevated carbon monoxide levels increase cholesterol uptake by coronary arteries, and the associated hypoxia encourages endothelial damage and enhances the atherosclerotic process. Smoking is strongly related to low levels of high density lipoprotein cholesterol, a factor in promoting intraarterial cholesterol accumulation. Hypertension combined with smoking markedly increases cardiovascular mortality in uremic patients. Other factors involved in endothelial cell damage and cholesterol accumulation, platelet activation and adherence, and smooth muscle cell proliferation are also discussed.

84–0816. Hillis, L. D.; Hirsh, P. D.; Campbell, W. B.; Firth, B. G. **Interactions of the Arterial Wall, Plaque, and Platelets in Myocardial Ischemia and Infarction.** *Cardiovascular Clinics* 14(1): 31-44, 1983.

Smoking as an initiator of atherosclerosis is discussed in a review of the roles of platelets, thromboxane, and prostacyclin in ischemic heart disease. There is some evidence that smoking causes a thromboxane—prostacyclin imbalance. It increases platelet activity and proaggregatory prostaglandin production, and it potentiates the effects of hyperlipidemia on platelet aggregability. Nicotine inhibits the ability of the coronary arteries to synthesize prostacyclin-like substances in vitro, an effect that is more pronounced in vascular tissue from individuals who smoke than from nonsmoking controls. The functions of platelets, thromboxane, and prostacyclin in the initiation, progression, and manifestation of coronary disease (including stable, unstable, and Prinzmetal's angina; acute myocardial infarction; and sudden death) are detailed. (Auth. Abs. Mod.)

84–0817. Kinlough-Rathbone, R. L.; Packham, M. A.; Mustard, J. F. **Vessel Injury, Platelet Adherence, and Platelet Survival.** *Arteriosclerosis* 3(6): 529-546, November-December 1983.

In discussing platelet survival in conditions in which vessel injury has been experimentally induced or occurs as a result of vascular disease, it is noted that although it is likely that some constituents of cigarette smoke damage vessel walls, the causative factor has not been identified. Nicotine, carbon monoxide, and tobacco antigen have all been suggested as being responsible. Shortened platelet survival has been observed in a number of clinical conditions that are associated with vessel injury, vascular grafts, and thromboembolism, but does not occur in all patients with these conditions. Although a number of drugs have been shown to prolong shortened platelet survival, in most cases this is not closely correlated with their effects on platelet aggregation and the release of the contents of platelet granules in vitro, nor with the clinical effects of the drugs. Drugs could influence platelet survival by reducing platelet consumption in thrombi, decreasing platelet adherence to the damaged vessel wall, or decreasing the rate of clearance of altered platelets by the reticuloendothelial system. (Auth. Abs. Mod.)

84–0818. Kraus, J. F.; Curb, J. D.; Cutter, G.; Daugherty, S. A.; Neill, K. C.; Wassertheil-Smoller, S. **Baseline Medical History Characteristics of the Hypertensive Participants.** *Hypertension* 5(6, Part 2): IV-51-IV-91, November-December 1983.

Data from the Hypertension Detection and Follow-Up Program (HDFP) showed that smoking rates decreased with age in a striking manner, were higher in men than women and in blacks than whites, and appeared to increase with increasing diastolic blood pressure. Questionnaires were completed by 10,940 hypertensive persons who were randomized into two treatment groups, Stepped Care and Referred Care, of the HDFP. Specific baseline information included history of cardiovascular events or conditions (such as myocardial infarction and intermittent claudication), respiratory symptoms and smoking, history of selected key neurological events and symptoms, history of other comorbid conditions, and family history of certain diseases. The prevalence of cigarette smoking was somewhat more common in those not taking antihypertensive medication at baseline. (Auth. Abs. Mod.)

84–0819. Kuznetsov, G. P.; Perepelkina, N. B. **Vliyaniye Kureniya na Nekotorye Pokazateli Kardiogemodinamiki u Bol'nykh so Stenokardiyey i Zdorovykh Lits. [The Effect of Smoking on Several Cardiohemodynamic Indicators in Angina Pectoris Patients and Healthy Persons.]** *Kardiologia* 11(22): 41-44, 1982, Russian.

The effects of smoking on several cardiohemodynamic parameters were investigated in 47 angina pectoris patients (22 smokers, 25 nonsmokers, average age 48±12 years) and 48 healthy controls (23 smokers, 25 nonsmokers, average age 27±7 years). Of the angina patients, 8 of the smokers and 15 of the nonsmokers had suffered myocardial infarctions. In healthy people, the increase in oxygen demand caused by smoking is partially compensated by an increase in coronary circulation. Under conditions of arterial sclerosis, the coronary arteries cannot adequately react to the stimulating effect of catecholamines, and the increased oxygen demand may lead to transitory ischemia. Tabular data on systolic and diastolic blood pressure, heart rate, and other electrocardiographic measures are provided.

84–0820. MacDougall, J. M.; Dembroski, T. M.; Slaats, S.; Herd, J. A.; Eliot, R. S. **Selective Cardiovascular Effects of Stress and Cigarette Smoking.** *Journal of Human Stress Research and Management* 9(3): 13-21, September 1983.

Subjects who smoked under stress showed markedly larger increases in all cardiovascular parameters measured, compared with smokers not under stress and sham smokers (with or without added stress). The 51 smokers were studied to determine how cigarette smoking and psychological stress combine to affect cardiovascular function. Stress was operationally defined as playing a series of difficult video games under challenging instructional conditions. Following an initial test game, 51 smokers were randomly assigned to a 2 (smoke vs. sham smoke) X 2 (stress vs. no stress) design. Subjects who sham smoked (inhaled unlit cigarettes) under no stress had minimal changes in cardiovascular parameters. Subjects who smoked under no stress evidenced approximately 12 mm Hg increase in systolic blood pressure and 9 mm Hg increase in diastolic blood pressure, and a 15 beat/minute increase in heart rate. These effects were similar in magnitude to those seen in subjects who sham smoked under stress. Values for smokers under stress were at least twice those produced by smoking or stress taken separately. Possible mechanisms of how smoking and stress may combine to heighten the risk of coronary disease are suggested. (Auth. Abs. Mod.)

84–0821. Meade, T. W. **Factor VII and Ischaemic Heart Disease: Epidemiological Evidence.** *Haemostasis* 13(3): 178-185, May-June 1983.

Factor VII appears to be unaffected by smoking, although cigarette use raises fibrinogen levels and lowers factor VIII levels. High levels of factor VII are associated with an increased risk of death from cardiovascular disease, especially ischemic heart disease (IHD). Theoretical, experimental, and epidemiological evidence all suggest that the association may be cause and effect: increasing age, diabetes, obesity, use of oral contraceptives, and occurrence of menopause are each associated with raised factor VII levels and increased risk of IHD. Analysis of Northwick Park Heart Study subjects, however, showed factor VII levels of 106.7 percent among 809 smokers and 107.0 percent in 1,022 nonsmokers (not significant). Conversely, black ethnic group membership and vegetarianism, both apparently protective against IHD, are associated with low factor VII levels. (Auth. Abs. Mod.)

84–0822. Rao, V. K.; Morrison, W. A.; O'Brien, B. McC. **Effect of Nicotine on Blood Flow and Patency of Experimental Microvascular Anastomosis.** *Annals of Plastic Surgery* 11(3): 206-209, September 1983.

Although no thrombosis occurred in these experiments on the effect of nicotine on blood flow, decreased flow was noted in the saphenous flap in dogs. This slowing of flow should predispose to thrombosis, especially if other factors associated with vascular repair were suboptimal. Nicotine is known to have deleterious effects on the cardiovascular system, causing vasoconstriction and increased blood pressure. A drop in blood flow velocity in humans, endothelial damage in rats, and inhibition of platelet aggregation in rabbits' blood have all been shown experimentally. Two studies investigate the effects of nicotine on microvascular anastomotic patency: (1) electromagnetic blood flow recordings were made in the saphenous artery of the saphenous flap of dogs after intraarterial installation of nicotine, with decreased blood flow consistently recorded up to a maximum of 72 percent of normal flow; (2) rabbit femoral arteries were anastomosed and patency rates at 3 weeks checked after various combinations of pre- and postoperative intraperitoneal nicotine had been injected. There was no significant increase in thrombosis between the groups treated with nicotine and controls. Results of this study, and the observed clinical effects in two patients with microvascular transfer, suggest that smoking in the perioperative period should be avoided. (Auth. Abs. Mod.)

84–0823. Rhomberg, H. P. **Koronare Risikofaktoren und Deren Beeinflussbarkeit auf Gemeindeebene. [Coronary Risk Factor Reduction on a Community Basis.]** *Fortschritte der Medizin* 101(42): 1935-1936, 1938, November 10, 1983, German.

A 1977 survey of coronary risk factors in the Tyrolean village of Oberperfuss revealed that 43.5 percent of the men (n=295) and 19.6 percent of the women (n=316) were smokers. Participants (70 to 75 percent of population) were between 20 and 64 years of age. Also, 11.2 percent of men and 14.9 percent of women had plasma cholesterol levels of ≥7.24 mmol/l. Incidence of hypertension was 122/1,000 men and 209/1,000 women. A community-oriented intervention program of health education, diet, and hypertension control medication was implemented with the help of local general practitioners. At 3-year followup, 9.3 percent of the male smokers and 9.6 percent of the female smokers had quit. Among those who continued to smoke, there was a small, but not significant, reduction in nicotine consumption. Men also showed a 23 percent reduction in cholesterol level, and a third of the hypertensives had normal blood pressure. Among the women, cholesterol levels dropped by 24 percent and half of the hypertensives had blood pressure under control. (Auth. Abs. Mod.)

84–0824. Rossouw, J. E.; Du Plessis, J. P.; Benade, A.J.S.; Jordaan, P.C.J.; Kotze, J. P.; Jooste, P. L.; Ferreira, J. J. **Coronary Risk Factor Screening in Three Rural Communities. The CORIS Baseline Study.** *South African Medical Journal* 64(12): 430-436, September 17, 1983.

A three-community survey for ischemic heart disease (IHD) among rural Afrikaans-speaking whites in the southwestern Cape revealed a high incidence of smoking, hypertension, and hypercholesteremia, the major reversible risk factors. In addition, minor factors such as inactivity, obesity, hyperuricemia, coronary-prone behavior and the irreversible risk factors of chest pain, ischemic changes on ECG, and a family history of IHD were common. Smoking was twice as common and daily consumption higher among males compared with females. Smoking prevalence in both sexes was highest between the ages of 35 and 44 (50.2 percent of males and 23.4 percent of females). Singly, or in combination, the major risk factors were present in the great majority of the study population after age 44. The interaction of high levels of lifestyle-induced risk factors with constitutional predisposition could adequately explain any excess risk of IHD in the Afrikaans-speaking community. (Auth. Abs. Mod.)

84–0825. Rowland, M.; Fulwood, R.; Kleinman, J. C. **Changes in Heart Disease Risk Factors.** In: Golden, R. M.; Wilson, R. W.; Karet, J. (Editors) *Health, United States, 1983, and Prevention Profile.* U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, DHHS Publication No. (PHS) 84-1232, December 1983, pp. 13-17.

Data from the National Health and Nutrition Examination Surveys (1971-1975 and 1976-1980) indicate that the proportion of adults who were current smokers decreased among black women (24 percent) and white and black men (7 and 5 percent, respectively); however, there was no change in smoking rates among white women. Since smoking, high blood pressure, and elevated cholesterol level have been identified as major controllable risk factors for coronary heart disease (CHD), the study focused on the changes in prevalence among adults aged 35 to 74, and any associated risk reduction. Expected CHD mortality declined by 13 to 16 percent for blacks and by 7 to 8 percent for whites (differences between men and women were not statistically significant). People with more than one elevated risk factor are at especially high risk of CHD mortality. Among blacks, who have the higher proportion of adults with two or more factors, the reduction has been greater than among whites, for elevated blood pressure and smoking. Results suggest that the reduction in risk factor levels could account for a substantial portion of the decline in CHD mortality, especially among black people. It is noted that the proportion of heavy smokers (≥25 cigarettes/day) did not change significantly; thus, the decline in smoking resulted from a reduction in the proportion of light and moderate smokers. Problems in interpreting the results from risk factor intervention trials are discussed.

84–0826. Shionoya, S. **What Is Buerger's Disease?** *World Journal of Surgery* 7(4): 544-551, July 1983.

While the etiology of Buerger's disease remains unknown, observations of 115 patients (113 males, 2 females) over a 4-year period at the Nagoya University Branch Hospital confirm a relationship with cigarette smoking. The disease, primarily of the small- and medium-sized vessels of the extremities, was arrested in patients who stopped smoking, but progressed in patients who continued or resumed smoking. All patients had been heavy smokers; age at onset of disease symptoms ranged from 19 to 49 years (average 35.9±7.6 years). In addition to smoking, diagnostic criteria included onset before 50 years, infrapopliteal arterial inclusion, phlebitis migrans or involvement of the upper extremity, and absence of atherogenic risk factors. From the time of onset, ulceration or gangrene occurred in 85 patients (73.9 percent), phlebitis migrans in 52 patients (45.2 percent), and involvement of the upper extremity in 94 patients (84.3 percent). Approximately 75 percent of all the patients had three- or four-limb involvement. Patency of bypass grafting, performed in 20 patients, was generally sufficient for healing of skin lesions. If the revascularized segment occluded after the ulceration had healed, trophic lesions did not recur unless the patient resumed smoking. Of patients who continued smoking despite warnings, four underwent major amputation.

84–0827. Wabrek, A. J.; Shelley, M. M.; Horowitz, L. M.; Bastarache, M. M.; Giuca, J. E. **Noninvasive Penile Arterial Evaluation in 120 Males With Erectile Dysfunction.** *Urology* 22(3): 230-234, September 1983.

Although the prevalence of smokers among the 120 males referred to a hospital-based Medical Sexology Program for evaluation of erectile dysfunction was higher than indicated in the most recent Surgeon General's report on successive male birth cohorts, there was no significant difference in the number of current smokers in each of the three penile brachial index (PBI) groups. Subjects had, as part of the evaluation, a noninvasive penile arterial assessment that included determination of (1) blood pressure in each of the six penile arteries, (2) patency of each cavernosal artery, and (3) brachial blood pressure. The systolic pressure from each artery was averaged and divided by the brachial systolic pressure to determine the PBI. When a PBI of 0.75 or less was correlated with a history of myocardial infarction, coronary artery bypass, or cerebral vascular accident, a p value of 0.069 resulted, certainly suggesting that each is a manifestation of arterial disease.

84–0828. Yarnell, J.W.G.; Fehily, A. M.; Milbank, J.; Kubicki, A. J.; Eastham, R.; Hayes, T. M. **Determinants of Plasma Lipoproteins and Coagulation Factors in Men From Caerphilly, South Wales.** *Journal of Epidemiology and Community Health* 37(2): 137-140, June 1983.

Smoking and dietary variables were significantly associated with plasma lipid concentrations (n=670) and coagulation factors (n=658), indicating the possibility of dietary intervention to influence plasma concentrations of major risk factors for ischemic heart disease. Data from the cross-sectional study of 711 men (aged 30 to 69) found that smoking and five additional factors were significantly associated with plasma concentrations of high density lipoprotein cholesterol, explaining 12.6 percent of the variance. Smoking was significantly associated with low density lipoprotein cholesterol and triglyceride, but not with total cholesterol; percentages explained by significantly associated variables were 8.4, 18.5, and 7.5, respectively. Chemical fibrinogen was associated principally with age and smoking habit, but body mass index, alcohol consumption, and cereal fiber contributed a further 2.9 percent of the variance. Clotting fibrinogen was significantly associated only with age and smoking habit. Viscosity was similarly associated principally with age, body mass index, and smoking habit. Factor VII levels were significantly, and only weakly, associated with age.

See also, **84–0723**

PREGNANCY AND INFANT HEALTH

84–0829. Bachman, J. W. **Prenatal Care of the Normal Pregnant Woman.** *Primary Care* 10(2): 145-160, June 1983.

In a discussion of the obstetrician's role and responsibilities in the prenatal care of the normal pregnant woman, smoking is included in the area of anticipatory counseling. It is recommended that physicians follow the guidelines of the Massachusetts Public Health Department, which are: (1) the initial prenatal visit includes a discussion of smoking and adverse effects, stressing that all unnecessary drugs should be avoided during pregnancy and that cigarette smoke contains over 1,000 drugs; (2) the risk is higher for smokers with previous perinatal loss leading to placental complications and anemia, or for those in the older age group; (3) any pregnant woman who has bleeding should be questioned about smoking, and smoking-related hazards should be reemphasized to smokers; (4) the physician should be able to provide smoking cessation information in the form of pamphlets, self-help groups, or community resources; and (5) smoking in the physician's office and patient areas should be prohibited and abstinence enforced. In addition to routine prenatal care, risk assessment and anticipatory counseling in other areas, including nutrition, alcohol consumption, caffeine, and drugs, are discussed.

84–0830. Caan, B.; Petitti, D. B.; Allen, L. H.; Picone, T. A. **Food Intake and Weight Gain in Pregnant Women. (Letters).** *American Journal of Clinical Nutrition* 38(5): 815-817, November 1983.

Response to a study of pregnancy outcome in North American women (*American Journal of Clinical Nutrition* 36(6): 1205-1213, December 1982) suggests that one of the most important findings may be that smoking and psychological stress may modify the efficiency of energy utilization for pregnancy weight gain. Since dietary energy intake explained only 20 percent of the variance in the weight gain of nonsmokers, it may also be assumed that individual variability in energy utilization efficiency substantially contributes to individual differences in pregnancy weight gain. It is recommended that the mechanism that accounts for differences in caloric utilization and of factors other than smoking and stress that affect caloric utilization be investigated. See also 83-0896.

84–0831. Greenland, S.; Richwald, G. A.; Honda, G. D. **The Effects of Marijuana Use During Pregnancy. II. A Study in a Low-Risk Home-Delivery Population.** *Drug and Alcohol Dependence* 11(3/4): 359-366, June 1983.

Rates of dysfunctional labor, precipitate labor, and meconium staining for marihuana users (n=41, 17 percent cigarette smokers) vs. nonusers (n=272, 11 percent cigarette smokers) were 43 vs. 35 percent, 13 vs. 8 percent, and 17 vs. 13 percent, respectively. Results, which were little changed after adjustment for confounding factors (alcohol consumption, cigarette smoking, parity, income), indicate that marihuana use near term may elevate risk of abnormal labor and meconium staining.

84–0832. Kern, K. **Wplyw Palenia Papierosow na Niska Mase Urodzeniowa Plodow i Rozwoj Dzieci. [Influence of Cigarette Smoking on Fetal Low Birth Weight and Newborns Development.]** *Ginekologia Polska* 54(7): 483-486, 1983, Polish.

The close relationship between cigarette smoking by pregnant women and the low birth weight in offspring is discussed, noting that weight gain during pregnancy is no different for smokers than for nonsmokers. The lower birth weight of fetuses from smoking mothers is proportional to the number of cigarettes smoked and is not dependent on other factors (e.g., genetic). Placental index can be used as a measurable exponent of the influence of smoking on birth weight. It is also noted that smoking during pregnancy influences the infant's development in the first years of life. (Auth. Abs.)

84–0833. Klink, F.; Jungblut, J. R.; Oberheuser, F.; Siegers, C. P. **Cadmium- und Bleikonzentrationen im Fruchtwasser von Rauchenden und Nichtrauchenden Gravida. [The Concentrations of Cadmium and Lead in the Amniotic Fluid of Pregnant Women Who Smoke and Who Do Not Smoke.]** *Geburtsh und Frauenheilkunde* 11(43): 695-698, November 1983, German.

Results of amniotic fluid examinations of smoking and nonsmoking women demonstrated no placental barrier to cadmium, since a direct correlation between cigarette smoking and cadmium concentration in amniotic fluid was found. Many studies have shown the unhealthy effects of smoking on the fetus, and several possible mechanisms for such effects are discussed. In animal experiments, high doses of cadmium resulted in mutations and teratogenicity, while low doses caused retardation of fetal growth. Cadmium and lead content of amniotic fluid did not correlate with gestational age, pregnancy disturbances, or the incidence of abortions. Lead concentration of smokers and nonsmokers were not significantly different; the small amounts of lead in cigarettes do not increase lead levels in amniotic fluid. (Auth. Abs.)

84–0834. Lehtovirta, P.; Forss, M.; Rauramo, I.; Kariniemi, V. **Acute Effects of Nicotine on Fetal Heart Rate Variability.** *British Journal of Obstetrics and Gynaecology* 90(8): 710-715, August 1983.

The effects of nicotine on fetal heart rate (FHR) variability were studied in seven women in the second trimester and eight women in the third trimester of pregnancy by giving them nicotine-containing chewing gum. The possible effects of carbon monoxide or some other agents of nicotine-free smoke on FHR variability were tested in eight women in the second and eight in the third trimester of pregnancy by giving them herbal cigarettes to smoke. The gum caused an acute decrease in the interval index of FHR variability in both trimesters, as has been observed previously in association with tobacco smoking, and an acute decrease in the differential index in the second trimester, but not in the third. These effects differ from those associated with tobacco smoking, during which the differential index decreased in the third trimester but remained unchanged in the second. In the second trimester the gum elevated the baseline FHR, as did tobacco smoking. In the third trimester, the gum decreased the baseline FHR in contrast to tobacco smoking, which had no effect on it. Unlike nicotine, smoking a herbal cigarette had no effect on FHR and no depressant effect on FHR variability. The only fetal response was a transient increase in the interval index 5 to 10 min after smoking in the second trimester. It is concluded that nicotine seems to be responsible for the depressant effect of tobacco smoke on the fetus, as manifested in lowered interval indices. The different responses of the differential index and baseline FHR to tobacco smoking and chewing nicotine-containing gum at different stages of gestation remain unexplained. (Auth. Abs.)

84–0835. Linn, S.; Schoenbaum, S. G.; Monson, R. R.; Rosner, R.; Stubblefield, P. G.; Ryan, K. J. **The Association of Marijuana Use With Outcome of Pregnancy.** *American Journal of Public Health* 73(10): 1161-1164, October 1983.

Analysis of interview and medical record data from 12,424 women at time of delivery found that smoking was directly associated with marihuana use; 53.3 percent of daily marihuana users (n = 137) also smoked cigarettes. About 10 percent of all subjects reported marihuana use during pregnancy, the majority (7.1 percent) as only occasional users. Low birth weight, short gestation, and major malformations occurred more often among offspring of marihuana users. When logistic regression was used to control for demographic characteristics, habits, and medical history data, these relationships were not statistically significant. The odds ratio for the occurrence of major malformations among marihuana users was 1.36, higher than odds ratios for other exogenous variables. More data are needed to establish firmly or rule out an association between marihuana usage and major malformations. Until more information is available, women should be advised not to use marihuana during pregnancy. (Auth. Abs. Mod.)

84–0836. Lundberg, M. S.; Livingston, G. K. **Sister-Chromatid Exchange Frequency in Lymphocytes of Smoking and Nonsmoking Mothers and Their Newborn Infants.** *Mutation Research* 121(3/4): 241-246, September 1983.

While earlier reports have shown that cigarette smoking is associated with a significant elevation in the frequency of sister-chromatid exchanges (SCE) in human lymphocytes regardless of sex, results indicate that the effect of smoking does not carry over to the newborn children of mothers who smoke during pregnancy. There was no difference between SCE frequency of nine infants born to eight smoking mothers and six infants born to six nonsmoking mothers. The lack of a neonatal effect could be due to insufficient exposure, inability of metabolic products to cross the placenta, or the placental detoxification of genotoxic agents. Whatever the reason for the difference between mothers and infants, it seems unlikely that the sequellae of low birth weight and other adverse effects observed in infants born to smoking mothers are caused by chromosomal disturbance measurable by neonatal SCE frequency. Results are consistent with the hypothesis that toxic substances in tobacco smoke interact with chromosomal DNA of circulating lymphocytes. (Auth. Abs. Mod.)

84–0837. Mactutus, C. F.; Fechter, L. D. **Prenatal Exposure to Carbon Monoxide: Learning and Memory Deficits.** *Science* 223(4634): 409-411, January 27, 1984.

Chronic prenatal exposure to carbon monoxide (CO) demonstrated impaired learning retention in young adult rats as indexed by reacquisition, resembling reported impairment in achievement scores noted during early childhood in the offspring of women who were heavy smokers during pregnancy. Exposing pregnant rats to 150 ppm CO produced only minor reductions in birth weight of the pups and gave no evidence of overt teratogenesis. However, behavioral evaluation of learning and memory processes in a two-way avoidance task suggested a functional deficit in the central nervous system of the exposed offspring. Multiple dependent measures and specific control groups confirmed that this deficit was independent of nonassociative or motivational alterations. (Auth. Abs. Mod.)

84–0838. Marbury, M. C.; Linn, S.; Monson, R. R.; Schoenbaum, S. C.; Stubblefield, P. G.; Ryan, K. J. **The Association of Alcohol Consumption With Outcome of Pregnancy.** *American Journal of Public Health* 73(10): 1165-1168, October 1983.

Data do not support an association between alcohol intake of two or more drinks/day and a decrease in birth weight that is independent of smoking. Of the 12,440 pregnant women interviewed at time of delivery, only 92 (0.7 percent) reported consuming 14 or more drinks/week; 50 percent of these were also smokers. Birth weight ranged from an average of 3353 g for those who did not drink or smoke, to an average of only 2919 g for heavy smokers in the highest alcohol consumption category. Within each group of alcohol use, women who smoked > 10 cigarettes/day had babies that weighed 250 g less than babies of nonsmokers. In the crude data, alcohol intake of 14 or more drinks/week was associated with a variety of adverse pregnancy outcomes, including low birth weight, gestational age under 37 weeks, stillbirth, and placenta abruptio. After use of logistic regression to control for confounding by demographic characteristics, smoking, parity and obstetric history, only the association of placenta abruptio with alcohol consumption of 14 or more drinks/week remained statistically significant. With the exception of placenta abruptio, alcohol intake of fewer than 14 drinks/week was not associated with an increased risk of any adverse outcome. No association was seen with congenital malformations at any level of alcohol intake. (Auth. Abs. Mod.)

84–0839. Martin, J. C.; Martin, D. C.; Sigman, G.; Day-Pfeiffer, H. **Saccharin Preferences in Food Deprived Aging Rats Are Altered as a Function of Perinatal Drug Exposure.** *Physiology and Behavior* 30(6): 853-858, June 1983.

Maternal exposure to central nervous system (CNS) activators (i.e., nicotine) during the gestational and nursing periods may exert a lasting influence on offspring taste preferences in rats. Changes in preference for concentrations of saccharin solutions were measured to determine whether perinatal exposure to CNS-activating drugs resulted in early and/or altered onset of aging. Primiparous rats were injected with nicotine or methamphetamine during the gestational and nursing periods. The preference of male offspring for various concentrations of saccharin in water, or plain water, was determined from the ages of 15 to 36 months. The pattern of preference for saccharin concentration and the pattern of total fluid consumption changed over the lifespan as a function of perinatal treatment in rats maintained at 80 percent of their free-feeding weights. Further shifts in preference were due to the maternal drug treatments. The drug-related shifts in preference over time were complex, indicating that the effects of maternal treatment are neither simple nor readily predictable. Significance for aging organisms is discussed. (Auth. Abs. Mod.)

84-0840. Mau, G.; Brinkmann, G.; Anger, H. **Lipoprotein Pattern in Umbilical Cord Blood of Neonates of Smoking and Non Smoking Mothers.** *Acta Paediatrica Scandinavica* 72(5): 755-756, September 1983.

The lipoprotein levels in umbilical cord blood from 78 neonates did not reflect any influence of maternal smoking during pregnancy. The subjects were divided according to consumption; 23 were nonsmokers, 10 mothers had quit when the pregnancy was known, 25 had smoked 5 to 15 cigarettes/day throughout the pregnancy, and 20 had smoked >15/day during pregnancy. Lipoprotein levels did not differ between the four groups. It is suggested that smoking-related changes in umbilical cord vessels are not correlated to arteriosclerosis in adults.

84-0841. McNeil, T. F.; Kaij, L.; Malmquist-Larsson, A. **Pregnant Women With Nonorganic Psychosis: Life Situation and Experience of Pregnancy.** *Acta Psychiatrica Scandinavica* 68(6): 445-457, December 1983.

Data from 88 women with a history of nonorganic psychosis and 104 demographically similar control women showed that there were more nonsmokers among the controls (55 percent) than the cases (41 percent). Also, considerably more cases (34 percent) than controls (12 percent) smoked >10 cigarettes/day, and an increased frequency of mothers smoking >10 cigarettes/day was observed in every diagnostic group when compared with controls. The women were interviewed during pregnancy after quickening concerning their experience of pregnancy and current life situation. The cases, compared with controls, also reported a significantly increased frequency of material-situational problems; interpersonal difficulties and lack of support from spouses and parents; initially unplanned and undesired pregnancies; negative attitudes toward the pregnancy after quickening; nervousness and anxiety; panic about delivery; and great concern for personal mental health in the near future. No significant difference between groups was found on frequency of typical pregnancy symptoms, the experienced effect of pregnancy on physical health, or concerns about the child's health or mothering. (Auth. Abs. Mod.)

84-0842. Mitchell, J. A.; Hammer, R. E.; Goldman, H. **Concomitant Reduction in Uterine Blood Flow and Intrauterine Oxygen Tension in the Rat Following Nicotine Administration.** *Advances in Experimental Medicine and Biology* 159: 231-241, 1983.

Nicotine, injected into pregnant mice, suppressed whole uterine blood flow and luminal oxygen concentration, decreased perfusion at the implantation site, and retarded growth of the nidus. Pregnant, pseudopregnant and control rats, injected with nicotine (5 mg/kg), were measured for oxygen tension, blood flow (whole uterus and implantation sites), cardiac output, and endometrial vascular permeability. Mean uterine perfusion was significantly reduced ($p < 0.005$ at 45 and 90 min following nicotine injection), cardiac output was reduced, and intrauterine oxygen tension markedly reduced. At implantation sites, blood flow was approximately twice as great as at adjacent areas, and after nicotine injection underwent a marked and sustained decrease at implantation sites, which weighed significantly less in treated vs. control animals. Nonimplantation sites were notably less responsive; blood flow was significantly reduced only briefly at 60 min. Cardiac output and endometrial vascular permeability were not altered. While the nicotine dose used greatly exceeded that encountered during smoking, it is concluded that since even a single inhalation of cigarette smoke can induce uterine vascular constriction and signs of fetal distress, the results may be important in understanding the effects of nicotine on human pregnancy.

84-0843. Prager, K.; Malin, H.; Graves, C.; Spiegler, D.; Richards, L.; Placek, P. **Maternal Smoking and Drinking Behavior Before and During Pregnancy.** In: Golden, P. M.; Wilson, R. W.; Kavet, J. (Editors). *Health, United States, 1983, and Prevention Profile* Washington, D.C., U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, DHHS Publication No. (PHS) 84-1232, December 1983, pp. 33-45.

Of 4,405 married women who returned mailed questionnaires on smoking and drinking habits (response rate 56 percent), nearly half of those under age 20 were smokers, compared with one of four among those age 25 or more. Prior to pregnancy, almost a third of the white, a fourth of the black, but less than a fourth of the Hispanic and other women smoked cigarettes. Among white women, 16 percent of those with a college education vs. 60 percent of those who had not finished high school were smokers. Conversely, alcohol consumption increased with age and education. During pregnancy, mothers were more likely to stop drinking than to quit smoking. Overall, 22 percent of the white, 14 percent of the black, and 13 percent of the other women both smoked and consumed alcohol during pregnancy. No differences in age or race were noted among those who quit smoking. However, only 10 percent of the women with minimal education compared with 24 percent with the most education quit smoking. Overall, 20 percent became abstinent, 27 percent reduced consumption, and the prevalence of those smoking ≥ 10 cigarettes/day dropped to 14 percent. Among white mothers, 22 percent both smoked and drank, compared with 12 to 14 percent of the others.

84–0844. Rush, D.; Cassano, P. **Relationship of Cigarette Smoking and Social Class to Birth Weight and Perinatal Mortality Among All Births in Britain, 5—11 April 1970.** *Journal of Epidemiology and Community Health* 37(4): 249-255, December 1983.

Survey results show that of the 16,688 women who delivered singletons, 41.3 percent reported smoking at term, with rates differing markedly by social class (15.4 percent smoking >5 cigarettes/day in class I vs. 46.0 percent in class V). Infants of nonsmokers weighed an average of 215 g more than infants of mothers who smoked ≥ 15 cigarettes/day. Only 43 percent of women in the lowest class (V) began pregnancy as nonsmokers. Of those who quit during pregnancy, the majority stopped between the second and eighth month. Infants of those who quit prior to the ninth month had birth weights similar to infants of nonsmokers. Little difference was noted in the birthweight decrement associated with smoking across class. Perinatal mortality, however, was increased only among smokers in the manual labor social classes. Although the offspring of more privileged smokers were not protected from intrauterine growth retardation, they did not suffer from increased perinatal mortality. Observations of other populations suggest a possible nutritional mediation of this protective effect. (Auth. Abs. Mod.)

84–0845. Sexton, M.; Hebel, J. R. **A Clinical Trial of Change in Maternal Smoking and Its Effect on Birth Weight.** *Journal of the American Medical Association* 251(7): 911-915, February 17, 1984.

Results from a prospective, randomized, and controlled experiment with 935 pregnant smokers demonstrate that a reduction of smoking during pregnancy improves the birth weight of the infant. The women were randomly assigned to either a control group or a treatment group that received assistance in smoking cessation through personal visits, telephone calls, and mail contacts. The program provided encouragement through information, support, practical guidance, and behavioral strategies. In the eighth month of pregnancy, differences between the two groups in salivary thiocyanate level and reported smoking were statistically significant. For single, live births, the treatment group infants were an average of 92 g heavier and were 0.6 cm longer than the control group infants. The decrement in weight related to smoking cannot be fully explained by gestational age. Findings suggest that some fetal growth retardation can be overcome by providing antismoking assistance to pregnant women. (Auth. Abs. Mod.)

84–0846. Siegers, C.-P.; Jungblut, J. R.; Klink, F.; Oberheuser, F. **Effect of Smoking on Cadmium and Lead Concentrations in Human Amniotic Fluid.** *Toxicology Letters* 19(3): 327-331, December 1983.

Analysis of the amniotic cadmium (Cd) and lead (Pb) concentrations in 155 pregnant women (128 nonsmokers and 27 smokers) showed a correlation between daily cigarette consumption and Cd levels, which were 2.58 ± 1.36 ng/l and 7.29 ± 2.39 ng/l in nonsmokers and smokers, respectively. Pb levels in nonsmokers and smokers were 23.98 ± 9.41 ng/l and 21.53 ± 7.16 ng/l, respectively. It is suggested that Cd accumulation in the placenta may impair its function and thus be embryotoxic, and that fetal growth retardation and weight loss may be due to smoking-related carbon monoxide and Cd-induced alterations in placental function. Amniotic Cd or Pb concentrations were not correlated with age, duration of pregnancy, blood pressure, or disorders of pregnancy.

84–0847. Simberg, N.; Hartiala, J.; Hietanen, E. **Development of the Lipolytic Activity in Isolated Perfused Perinatal Rabbit Lungs and the Influence of Maternal Cigarette Smoke Exposure on It.** *Biology of the Neonate* 44(5): 264-271, November 1983.

The exposure of pregnant rabbits to cigarette smoke during the last 10 days before delivery did not affect the pulmonary lipolytic activity of the offspring. The development of the lipolytic activity of lungs obtained from late fetal and neonatal rabbits was studied and results correlated to the phospholipid content of lungs of similar ages. Isolated lungs were perfused and the release of fatty acids into the perfusion medium was analyzed by both colorimetric and radiochemical methods. The phospholipids of the postmitochondrial supernatant fractions of the lungs were extracted and quantified by measuring inorganic phosphorus. A high lipolytic activity in the lungs of 28-day-old fetuses was detected. The activity decreased toward birth, and was lowest on the first day after birth (about 20 percent of that observed in 28-day-old fetuses). However, it increased again during the first week after birth. It is concluded that changes in lung lipolytic activity influence lung phospholipid synthesis, and consequently also influencing surfactant formation in the lungs. (Auth. Abs. Mod.)

84–0848. Stein, Z.; Kline, J. **Smoking, Alcohol and Reproduction.** *American Journal of Public Health* 73(10): 1154-1156, October 1983.

With an overwhelming consistency across studies, smoking during pregnancy is associated with a reduction in birth weight of 150 to 250 grams, and a decrease in mean gestation by 1 to 2 days. This association has persisted over 20 years despite the reported reductions in tar and nicotine yields. Also, not all of smoking's consequences (including spontaneous abortion) can be correlated to maternal consumption during the intrauterine period, raising questions concerning the cumulative effects of smoking prior to pregnancy. Compared with the smoking data, the literature on the effects of alcohol consumption during pregnancy are disturbingly inconsistent. Many observations support the view that the effects of alcohol are independent of those of tobacco. However, much of the alcohol research failed to take smoking habits into account, which is considered as one of several factors contributing to the inconsistency.

See also, **84–0708, 84–0911**

OTHER DISEASES AND CONDITIONS

84–0849. Baron, J. A. **Smoking and Estrogen-Related Disease.** *American Journal of Epidemiology* 119(1): 9-22, January 1984.

In this review of the relationship of smoking with estrogen-related disease, epidemiologic data strongly support an association with an early natural menopause. There is also suggestive evidence that cigarettes may influence the risks of osteoporosis and endometrial cancer in ways not explained simply by the early cessation of menses in female smokers. The menopause risks of ex-smokers have regularly been found to be closer to those of never smokers than to those of current smokers, suggesting that the influence of smoking is reversible. Ex-smokers may also be at a higher risk of breast cancer than current smokers, perhaps also reflecting hormonal changes associated with cessation of smoking. However, the precise role of estrogens in breast cancer etiology remains undefined. Other smoking-related data on oral contraceptive use and hormone replacement therapy are discussed, as are the effects of smoking on ovarian function. Further investigation is required to substantiate the impact of smoking on estrogenic processes.

84–0850. Braganza, J. M. **Hypothesis. Pancreatic Disease: A Casualty of Hepatic "Detoxification"?** *Lancet* 2(8357): 1000-1003, October 29, 1983.

Inducers of the hepatic mixed-function oxidase (MFO) system (cigarettes, drugs, alcohol, coffee) are suspected factors in pancreatic disease, a suggested result of aberrant function of MFOs. The degree of inducibility is governed by host factors, including genetic endowment, and induction is facilitated by an ample supply of dietary polyunsaturated fatty acids. It is suggested that the products of hepatic detoxification (lipid peroxidation products, toxic epoxides, carcinogens, free radicals) are excreted in bile, reflux into the pancreatic duct, and induce pathological changes. Factors that promote reflux would increase risk of disease only when bile contains excessive amounts or abnormal types of reactive intermediates. (Auth. Abs. Mod.)

84–0851. Classen, M.; Hagenmuller, F. **Effect of Sucralfate on Peptic Ulcer Recurrence: A Controlled Double-Blind Multicenter Study.** *Gastroenterology* 18(Supplement 83): 61-68, 1983.

Data from a double-blind, placebo-controlled, 6-month trial on the efficacy of sucralfate for preventing ulcer recurrence show that there was a higher relapse rate among smokers compared with nonsmokers in the sucralfate and the placebo groups, although the differences were not significant. Participants, 174 patients (76 smokers) with healed duodenal ulcer and 77 (33 smokers) with healed gastric ulcer, received either 1 g sucralfate or placebo twice daily. The relapse rate at 6 months was 21.2 percent for sucralfate and 50 percent for placebo in the duodenal ulcer group; no significant difference in relapse rate was found in the gastric ulcer group. Both treatments were well tolerated. Two patients in each treatment group complained of nausea and epigastric pain immediately after drug intake, but no other drug-related symptoms were observed. It is concluded that sucralfate is effective in the prophylaxis of duodenal ulcer. No significant effect was found in the prevention of gastric ulcer recurrence. (Auth. Abs. Mod.)

84–0852. Donaghy, M.; Rees, A. J. **Cigarette Smoking and Lung Haemorrhage in Glomerulonephritis Caused by Autoantibodies to Glomerular Basement Membrane.** *Lancet* 2(8364): 1390-1392, December 17, 1983.

Of 47 patients who provided a complete smoking history, all 37 cigarette smokers suffered lung hemorrhage compared with only 2 of 10 nonsmokers. The smoking habits of 51 patients with glomerulonephritis (43 with pulmonary hemorrhage, 8 without) caused by autoantibodies to the glomerular basement membrane (GBM) were investigated. There was no significant difference between the titers of circulating anti-GBM antibodies in smokers and nonsmokers. In one patient, resumption of smoking was closely followed by recrudescence of lung hemorrhage. Results largely explain the variable presence of pulmonary hemorrhage in anti-GBM disease. (Auth. Abs. Mod.)

84–0853. Douglass, C. W.; Gillings, D.; Sollecito, W.; Gammon, M. **National Trends in the Prevalence and Severity of the Periodontal Diseases.** *Journal of the American Dental Association* 107: 403-412, September 1983.

In comparing data from national health surveys conducted in 1960 to 1962 and 1971 to 1974, the downward trend in cigarette consumption for males and females is consistent with national trends in gingivitis incidence. The overall decline in gingivitis scores is more pronounced in males, which may reflect the sharper decline in smoking rates in males compared with females (21 percent vs. 10 percent, respectively) over the same period. Although not all studies have shown a positive association between cigarette smoking and periodontal disease scores, the weight of evidence would seem to suggest a relationship. The significance of other variables such as oral hygiene index scores, socioeconomic status, dental care utilization, fluorides, and antibiotics is also discussed. A hypothesis is proposed, linking the slight increase in the more severe signs of periodontal disease in older adults to an increase in the number of teeth at risk; both total loss of teeth and number of missing teeth per person decreased for all ages between the two survey periods. This trend may increase with the growth of the nation's older population.

84–0854. Feldman, R. S.; Bravacos, J. S.; Rose, C. L. **Association Between Smoking Different Tobacco Products and Periodontal Disease Indexes.** *Journal of Periodontology* 54(8): 481-487, August 1983.

The finding of significantly greater alveolar bone loss and significantly greater pocket depth in cigarette smokers compared with cigar/pipe smokers and nonsmokers suggests a tobacco-related effect in systemic physiologic action. Six indexes of periodontal health, recorded in cigarette smokers, pipe/cigar smokers, and nonsmokers from the Veterans Administration Dental Longitudinal Study, included calculus deposition, plaque accumulation, gingival inflammation, periodontal pocket depth, alveolar bone loss, and tooth mobility. Cigarette smokers had significantly more calculus deposition than pipe/cigar smokers, although both groups had more calculus than nonsmokers. Cigarette smokers accumulated slightly less plaque than pipe/cigar smokers, and both groups accumulated less plaque than nonsmokers. Gingival inflammation and tooth mobility did not differ between smokers and nonsmokers, or between the two smoker groups. After covariance adjustment for age and calculus, all smokers had similar peridontal pocket depth and less plaque, gingival inflammation, and tooth mobility than nonsmokers, but only cigarette smokers had greater bone loss. (Auth. Abs. Mod.)

84–0855. Greer, R. O., Jr.; Poulson, T. C. **Oral Tissue Alterations Associated With the Use of Smokeless Tobacco by Teen-agers. Part I. Clinical Findings.** *Oral Surgery, Oral Medicine Oral Pathology* 56(3): 275-284, September 1983.

Among 117 smokeless tobacco users (113 boys, 4 girls), from a sample of 1,119 high school students, 57 (48.7 percent) had soft-tissue lesions and/or periodontal inflammation or erosion of dental hard tissue. It was found that four distinct lesions associated with smokeless tobacco use could be clinically identified: (1) hyperkeratotic or erythroplakic lesions of the oral mucosa; (2) gingival or periodontal inflammation; (3) a combination of oral mucosal lesions and periodontal inflammation; and (4) cervical erosion of the teeth. Among the 117 users, 99 were Caucasian, 6 Hispanic, 1 black, 1 Asian, 1 American Indian, and 6 failed to identify an ethnic origin. Use ranged from 1 to 20 "dips"/day, with an average time per dip of 30 minutes. Most users had been dipping for an average of 2 years, and 12 different tobacco brands were identified. (Auth. Abs. Mod.)

84–0856. Holmstrup, P.; Bessermann, M. **Clinical, Therapeutic, and Pathogenic Aspects of Chronic Oral Multifocal Candidiasis.** *Oral Surgery Oral Medicine Oral Pathology* 56(4): 388-395, October 1983.

A total of 97 separate chronic oral multifocal candidiasis lesions were diagnosed in 32 patients (11 females, 21 males) who were all tobacco users; 21 were also denture wearers. Tobacco habits recorded showed 19 smoked cigarettes, 8 cheroots, and 11 pipe tobacco. Mean duration of the habits exceeded 26 years (range 15 to 50 years). Patients were treated with antimycotics, for an average of 46 days for denture wearers and 44 days for nondenture wearers. Three patients quit smoking, six reduced consumption 50 percent, and four changed their habits qualitatively during the study. The three who quit had six lesions, only one of which recurred. While the six who decreased consumption did not show a reduced recurrence rate, the recurrent lesions were smaller. Changing forms of tobacco consumption did not influence recurrence rate. The palatal lesions in nondenture wearers and nodular lesions of the commissural areas showed the highest recurrence rate after 6 and 12 months.

84–0857. Michielutte, R.; Diseker, R. A.; Corbett, W. T.; May, W. J. **Characteristics of Women With Cervical Intraepithelial Dysplasia.** *Progress in Clinical and Biological Research* 130: 203-215, 1983.

Results of a case—control study (199 cases, 195 controls) indicate that women who use cigarettes, alcohol, and drugs are at significant risk of acquiring severe cervical dysplasia. Participants were young (mean age 21), of low socioeconomic status, and most (70 percent) were black. Other significant risk factors found were menorrhagia, amenorrhea, use of anticoagulants, visual problems, asthma and allergies, migraine headaches, multiple sex partners without contraception, and nervous conditions. Prior health history (cumulative effect) was strongly associated. Use of oral contraceptives was found to be protective. No association was found for early age of onset of sexual activity. Further study of the relationship between smoking and dysplasia is suggested.

84–0858. Svensson, H.-O.; Vedin, A.; Wilhelmsson, C.; Andersson, G.B.J. **Low-Back Pain in Relation to Other Diseases and Cardiovascular Risk Factors.** *Spine* 8(3): 277-285, 1983.

There were more smokers among men with low back pain (LBP) than among controls, and the association between LBP and smoking persisted in covariance analysis ($p < 0.05$). Other variables directly associated with LBP were calf pain on exertion, job-associated physical activity, and frequent feelings of worry and tension. LBP relationship to other diseases and cardiovascular risk factors was studied in 940 randomly selected 40- to 47-year-old males (42.5 percent smokers, 23.2 percent ex-smokers, 34.2 percent nonsmokers). Among smokers, 27 percent consumed more than 15 g tobacco/day; the average duration, 25 years. Lifetime incidence of LBP was 61 percent, prevalence was 31 percent, and subjects with severe pain constituted 16 percent. Prevalence of smokers was higher among subjects who had experienced pain at some time, and those with ongoing pain at time of examination or LBP at least once/month ($p<0.01$); and tended to be higher among subjects with ongoing daily pain, or LBP at least twice/week ($p<0.10$). While there were no differences in total tobacco consumption between the groups, subjects with severe pain had been smoking for more years ($p<0.05$). Since a positive correlation between cigarette smoking and diminished mineral content of bone has been identified in recent years, osteoporosis-related microfractures of the trabeculae in the lumbar vertebral bodies may be a possible LBP cause.

84-0859. Van Wyk, C. W.; Ambrosio, S. C. **Leukoedema: Ultrastructural and Histochemical Observations.** *Journal of Oral Pathology* 12(5): 319-329, October 1983.

Data indicate that the quantity of tobacco smoked by 12 males with leukoedema differed significantly from the quantity smoked by those without leukoedema. The cases (aged 16 to 22) had smoked from 4 to 10 years, 8 to 40 g/day; controls had smoked 2 to 10 g/day for 4 to 10 years. The ultrastructural features were investigated and compared with clinically normal buccal mucosa from four male smokers (controls). Histochemistry resolved some of the observed ultrastructural changes. The characteristic intracellular edema of the epithelial cells in leukoedema is due to vacuolation in the cytoplasm of cells. It is suggested that the vacuolation represents a limited reversible form of cellular degeneration resulting from cell damage, and that impeded mitochondrial function may be the cause of the vacuolation. The superficial "ballooning" cells are degenerated cells. The flattening of the vacuolated cells into a compact layer and the presence of keratohyalin granules and keratohyalin-like structures in the superficial cells are regarded as features of an aborted form of keratinization. It appears that there is a threshold for smoking and, when it is exceeded, leukoedema may develop, but the mechanism by which smoking causes cell damage is not yet known. (Auth. Abs. Mod.)

84-0860. Vogt, T. M. **Effects of Parental Smoking on Medical Care Utilization by Children.** *American Journal of Public Health* 74(1): 30-34, January 1984.

Children ($n=767$) in nonsmoking households used significantly more outpatient services than did children ($n=839$) from households reporting at least one smoker; however, the greatest differences were in the number of well-child physicals performed. Information on smoking habits was from a household interview survey of 2,582 adult members of the Kaiser-Permanente Medical Care Program of Oregon. Detailed, computerized medical care records were maintained for all inpatient and outpatient care rendered between 1967 and 1974 to the 1,761 children (those over age 18 excluded) of the interviewed sample. When adjusted for age, family size, socioeconomic status, and duration of health plan membership, there were no significant differences in inpatient and outpatient medical care use for respiratory illness by children of smoking compared with nonsmoking households. Since studies have suggested that children in smoking households have higher rates of respiratory diseases, it appears that parents who smoke are less likely to seek care for mild outpatient illnesses, or take children in for preventive care. It is suggested that parents need to be educated not only about the risks of cigarette smoking, but also about the need for providing proper preventive and outpatient medical care for their children.

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

84-0861. Akgun, N.; Baser, E.; Ozsoy, N.; Yapicioglu, S. **Izmir'de Orta Ogretim Ogrencileri, Tip Ogrencileri ve Tip Fakultesi Ogretim Uyeleri ve Yardimcilari Arasinda Sigara Icimi. [Smoking Among Secondary School Children, Medical Students and Medical Teaching Staff of Ege University Medical School in Izmir, Turkey.]** *Turkish Journal of Sports Medicine* 18(4): 147-169, December 1983, Turkish.

Among 3,100 secondary students surveyed in 1981, 25.5 percent of girls and 37.7 percent of boys were smokers (consumed at least one cigarette/day or seven/week). Smoking rates among 660 medical students were 31.8 percent of females and 47 percent of males. Of the 254 medical teaching staff members, 52.5 percent of the females and 54 percent of the males were smokers. Since 1971, prevalence of cigarette use among female students had not changed significantly, although the proportion of heavy smokers had increased dramatically. Among male students, however, the prevalence of both light and heavy smoking had increased. Reasons for smoking cited by students included peer pressure, social acceptance, curiosity, and reduction of embarrassment. Parental cigarette use was not correlated to the students' habits. The age at onset was generally 15 for the secondary school students and 17 for the medical students. The majority smoked filtered cigarettes.

84-0862. Anderson, R. **How Have People Changed Their Health Behaviour?** *Health Education Journal* 42(3): 82-86, 1983.

Assessment of behavior changes that respondents (n = 822) considered health-related indicated that 10 percent had stopped or reduced smoking and 5 percent had started or increased smoking. Results are based on responses to a larger study of changes in attitudes and practices in Great Britain, conducted in 1977, which covered the previous 10-year period. Data, which reflect unprompted responses of subjects and are not derived from direct questioning, indicate that (1) smoking initiation was not related to sex or social class; (2) there was a decline from 19 percent of 18- to 24-year-olds to 1 percent of 55- to 64-year-olds in the proportion reporting starting smoking within the previous 10 years; and (3) no one aged 65 or over reported starting to smoke. The proportion reporting cessation of smoking was not associated with age, social class, or self-assessment of health, but 16 percent of the men had stopped smoking compared with 3 percent of women. Ninety percent of those who reported starting or increasing smoking thought they were less healthy as a result, and 91 percent who said they had stopped or reduced smoking believed they were more healthy. Frequency of consultation with doctors over the previous year was not associated with reports of giving up smoking or drinking.

84–0863. Bailey, A. **Fewer Connecticut High Schools Allowing Smoking.** *Connecticut Health Bulletin* 97(4): 280-281, 1983.

While a trend toward allowing student smoking in Connecticut high schools has reversed, none of the 163 public high schools surveyed prohibited cigarette use by faculty members. A continuing survey by two State health agencies shows that in January 1983, smoking was allowed in 75.5 percent of the State's public high schools, down 4.4 percent from a 1980 high of 79.8 percent. The 1980 figure capped a rise of 38.8 percent from 1972. Smoking in private high schools is now allowed at only 42.3 percent of those institutions, down from a high of 53.2 percent in 1974. Most of the schools that permitted smoking also carried restrictions, such as requiring parental permission, a minimum age or grade level, or only in certain locations. Private schools were more likely than public schools to have such eligibility requirements. The schools that banned smoking did not impose heavier punishments on violators than those that permitted smoking with certain restrictions. Typical punishment was suspension and/or detention, and some required parent-teacher conferences and other penalties. (Auth. Abs. Mod.)

84–0864. Barker, M. E.; Churms, B.; Henchy, M.; Hibble, A. G.; Mitchell, J. V.; Prasad, K. S.; Scarisbrick, C. **A General Practice Approach to Influencing Smoking Habits.** *Journal of the Royal College of General Practitioners* 33(254): 539, 541, September 1983.

General practitioners can play a crucial role in inducing smoking cessation among patients, and are encouraged to identify smokers and acquaint them with smoking hazards and available smoking cessation support. It is indicated that smoking cessation advice combined with a followup visit to assess progress could produce about 25 long-term successes annually, and participation by all general practitioners in the United Kingdom could yield more than one-half million ex-smokers each year. Physicians' antismoking efforts may be enhanced through emphasis on different advantages of nonsmoking based on category of patient, emphasis on social acceptability, anticipating questions and answers, preplanning the first day of smoking cessation, antismoking literature, and commercially available smoking cessation aids. General practitioners are advised to influence children's attitudes toward smoking through discussions, health education, and influencing the attitudes of parents.

84–0865. Bates, R. C. **Doctors Who Smoke.** *New York State Journal of Medicine* 83(13): 1294, December 1983.

While physicians' cigarette consumption is only about half that of the general population, those who smoke are discredited by issues that have medical, moral, and philosophic components. The doctor—smoker may be alienated from himself, since, with few exceptions, he is engaging in behavior that he cannot approve. Furthermore, he is marketing a product, health preservation, that he does not buy himself. To the extent that physicians are more influential and considered exemplars, their smoking and otherwise negative acts are more damaging to others and may communicate to laymen that rules regarding health are not important. Underlying all health-threatening behaviors is the magical belief in one's omnipotence, the single defense dangerously attractive to physicians. However, cigarettes, storms, heart attacks, cancer, and the laws of probability have no respect for medical degrees.

84–0866. Bennett, W. **The Nicotine Fix.** *Rhode Island Medical Journal* 66(11): 455-458, November 1983.

Since most smokers become addicted before age 20 (an adolescent who has smoked more than one cigarette has only a 15 percent chance of remaining a nonsmoker), the American tobacco industry has focused marketing efforts on minors throughout its phenomenal growth (from 2.5 billion cigarettes in 1889 to 704 billion in 1982). Five of the 10 most heavily advertised American products are cigarette brands, which are promoted on half the nation's billboards. Health, a long-dominant theme in cigarette advertising, continues to be an issue: low-tar brands have grown from 10 to 43 percent of the American market within the past 5 years. Paradoxically, low-tar brands must be smoked more heavily to prevent withdrawal, and the low-tar smoker can be expected to consume more cigarettes and circulate greater quantities of cigarette smoke (the major source of air pollution for nonsmokers). Cigarette smokers of both sexes carry an increased burden of disease throughout their lives; 54 million adult and 5 million adolescent smokers may never reach age 70. However, an end to cigarette addiction and the 17 billion dollar tobacco industry could bring economic disaster. Short-term gains (increased productivity and diminishing hospital costs) would disappear in 20 years due to a larger elderly population requiring social security payments and health care. The development of the American cigarette industry and the mechanisms and nature of nicotine addiction are also described.

84-0867. Bethwaite, J.; Coope, P.; Ashe, S.; Dunbar, R.; Hutchinson, M.; Jackson, D.; Johnston, A.; Lay, M.; Linton, T.; McKenzie, M.; Tennent, L.; Wilkinson, T. **Smoking at School. (Letter).** *New Zealand Medical Journal* 96(743, Part 2): 945-946, November 9, 1983.

Among New Zealand third form students (143 male, 130 female, mean age 13 years 7 months), 16 percent were smokers, 40 percent had tried smoking and given it up, and 44 percent had never smoked. The most frequent reasons given for quitting were (1) concern about health and fitness (75 percent of boys, 47 percent of girls); (2) satisfied curiosity (41 percent of boys, 63 percent of girls); (3) guilt feelings (39 percent of boys, 51 percent of girls); (4) concern about future health (51 percent of boys, 29 percent of girls); and (5) distaste for cigarette smell (38 percent of boys, 41 percent of girls). The sex differences were statistically significant. Among never smokers, over 90 percent cited health concerns as the reason. It is suggested that this information may be useful in formulating antismoking education and advertising programs.

84-0868. Blum, A. **Using Athletes to Push Tobacco to Children. Snuff-dippin' Cancer-Lipped Man.** *New York State Journal of Medicine* 83(13): 1365-1367, December 1983.

The sharp increase in sales of smokeless tobacco (snuff and chewing tobacco) since 1960 reflects the response of manufacturers to the public's concern about the health effects of smoking, especially cigarettes. Emphasis in advertising has shifted to smokeless types, since such promotion is not yet banned on television. An array of celebrities, mainly sports personalities, extol the virtues of the smokeless brands, creating an image for the young that is difficult to counteract. Related products (shirts and caps) keep the image prominently displayed while candy and chewing gum, packaged to look like tobacco items, influence even the youngest users with advertising slogans that suggest the relationship; e.g., a powdered bubble gum in a round tin carrying the slogan "Best chew this side of the Pecos." The United States Tobacco Company has created a special program for the 1984 Olympic games, and while many prominent sponsors have condemned drug abuse among athletes, none has publicly challenged the campaign of the tobacco company.

84-0869. Blum, A.; Rosenberg, J. **When "More Doctors Smoked Camels": Cigarette Advertising in the** ***Journal.*** *New York State Journal of Medicine* 83(13): 1347-1352, December 1983.

Current promotion of smoking, by generating complacency and tolerance among nonusers as well as sales to potential or current smokers, is even more pernicious than old promotions that generated acquiescence to advertising cigarettes in a medical context (i.e., "More doctors smoke Camels"). By ignoring cigarette advertising or failing to become angry, physicians, as leaders in society, are doing precisely as the tobacco manufacturers wish. Despite the attitude that smoking will gradually die out and the cigarette companies will diversify into other businesses, the cigarette companies (especially those aiming at developing nations) have other intentions. Removal of all economic incentives for cigarette companies and their subsidiaries is suggested, with a physician-led selective economic boycott as a possible first step. The increasing number of medical research councils, institutes, and science symposia underwritten by tobacco companies, and of medical and business schools that accept endowment money, indicate a growing possibility of tobacco industry ownership and control over health care services and providers. Cigarette advertising in the *New York State Journal of Medicine* is traced from 1933 to 1955, and advertising in the *Journal of the American Medical Association*, peer-reviewed medical journals, and throwaway medical magazines is discussed.

84-0870. Borkon, L.; Baird, D. M.; Siff, M. **Tobacco Smoking Among Students at the University of the Witwatersrand, Johannesburg.** *South African Medical Journal* 64(21): 809-812, November 12, 1983.

Data on 1,632 full-time college students showed that 23 percent of the males and 19 percent of the females were smokers, 197 males and 78 females classified themselves as ex-smokers, and 61 percent of the total sample were nonsmokers. Most smokers consumed between 10 and 20 cigarettes/day. The years between 17 and 19 appear to be important in determining whether the student became a regular smoker; 19 percent were regular smokers at age 18. Most ex-smokers also stopped between 17 and 19 years of age, suggesting that this age group is easily influenced. Only 8 percent stopped smoking when over 21 years of age. Motivating factors in initiation included (1) purely experimental reasons, (2) academic stress, (3) peer pressure and social conformity, (4) parental example, and (5) nonacademic stress. The main reasons for stopping smoking were a desire for physical fitness and the knowledge that smoking is a hazard to health. It is noted that 53.2 percent of the students continue to smoke because it is automatic (a habit), and for relaxation. It is recommended that the university health services coordinate or conduct regular rehabilitation courses for smokers on campus.

84-0871. Centers for Disease Control. **Behavioral Risk-Factor Prevalence Surveys — United States, Third Quarter 1982.** *Morbidity and Mortality Weekly Report* 32(46): 603-604, 609-610, November 25, 1983.

Data, from a seven-State questionnaire survey in the third quarter of 1982, show that in five States (Iowa, Michigan, New Mexico, Tennessee, Texas) there are more male smokers (ranging from 31.3 percent in New Mexico to 42.4 percent in Tennessee) than female smokers (23.8 percent to 30.2 percent).

However, female smokers outnumber male smokers in Arizona (33.3 percent vs. 28.3 percent) and in New Hampshire (41.3 percent vs. 29.8 percent). In general, results are consistent with other state-based behavioral risk factor surveys conducted during the first and second quarters of 1982, which reported that more men than women are smokers. Other consistent distinctions are: (1) alcohol misuse is more prevalent among men; (2) risk of hypertension increases with age; and (3) obesity prevalence increases to middle age and then declines. Regional distinctions include (1) uncontrolled hypertension is more apparent in the southeastern states surveyed; (2) alcohol misuse is lower in the southeastern states, except Florida; and (3) obesity is more prevalent in the eastern states.

84–0872. Chen, M. S., Jr.; Bill, D. **Statewide Survey of Risk Factor Prevalence: The Ohio Experience.** *Public Health Reports* 98(5): 443-448, September-October 1983.

A 1982 Ohio health risk factor survey ("Health Ohio") showed that 31 percent of the random population sample (n = 607, aged 18 and over) were current smokers, a proportion similar to data from Massachusetts and to national statistics. Smoking began between the ages of 14 and 18 years for 50 percent of all current smokers. Age of smoking initiation, compared for male vs. female respondents, was younger for males, some starting as early as 8 years of age. Additional health risk factors surveyed included alcohol consumption, hypertension, and accident prevention and injury control. Findings indicated the need to (1) remove economic barriers that impede the installation of residential smoke detectors; (2) initiate health education at an earlier age to counter cigarette smoking trends; and (3) encourage adult self-determination in reducing health risks. The survey data, describing the need for intervention to reduce health risk factors, have become the basis for health education—risk reduction efforts of the Ohio Department of Health. (Auth. Abs. Mod.)

84–0873. Chesterfield-Evans, A. **BUGA-UP (Billboard Utilizing Graffitists Against Unhealthy Promotions). An Australian Movement to End Cigarette Advertising.** *New York State Journal of Medicine* 83(13): 1333-1334, December 1983.

In a determined bid to eliminate cigarette advertising, BUGA-UP, a group that includes physicians, journalists, teachers, and clergymen, is spray-painting antismoking satiric messages on advertising billboards. Between 1980 and 1983, 38 member arrests have been made, but fines have been nominal. BUGA-UP members have entered a man who smoked through a tracheostomy tube in a "Marlboro Man of Australia" contest, been chained to a racing car decorated with Marlboro decals, publicized Amatil (British American Tobacco's Australian company) involvement with the Australian ballet, and opposed cigarette advertising at a children's fair and tobacco company sponsorship of sporting events such as the Australian Open Tennis Championships. Public support for the group has remained strong.

84–0874. Cohen, S. B. **Are Physicians to Blame If Patients Smoke?** *New York State Journal of Medicine* 83(13): 1295, December 1983.

While all physicians acknowledge that nonsmoking is desirable, they may be indifferent about taking responsibility for smoking in their offices or professional meetings, or in their patients' lives. Attitudes and behaviors that derive from society are relevant to the passivity of many physicians. They may advise patients not to smoke, but do not consider it their responsibility to help patients to quit or refer them for help unless the patients request it. Physicians are urged to reassure patients that, despite the difficulties encountered in quitting, ending dependence on cigarettes may be a most rewarding accomplishment. Furthermore, physicians concerned about creating smoke-free hospitals should not have to explain the cost-effectiveness and improved quality of the environment; rather, hospital administrators should explain why so little has been done.

84–0875. Covey, L. S.; Mushinski, M. H.; Wynder, E. L. **Smoking Habits in a Hospitalized Population: 1970—1980.** *American Journal of Public Health* 73(11): 1293-1297, November 1983.

Findings indicate the limited effectiveness of public health education regarding smoking in lower socioeconomic groups, and suggest that lung cancer and other diseases for which smoking is a major factor will become increasingly related to social class. The smoking habits of 23,953 hospitalized men and women aged 20 to 80 years, interviewed in nine United States cities between 1970 and 1980, were examined. Comparisons were made of age-adjusted smoking rates in 1970 to 1975 vs. 1976 to 1980, stratified by sex, race, and educational level. An overall decrease in smoking exposure between the time periods studied was observed: rates of current cigarette smokers declined and consumption of cigarettes containing ≤ 12 mg tar increased. Nevertheless, among current smokers, there was no reduction in the proportions of those who smoke 31 or more cigarettes daily. An enhancing effect of higher education on rates of smoking cessation and use of cigarettes containing ≤ 12 mg tar was observed. In both time periods, proportionately more black than white men were smokers, although the difference was smaller in college-educated groups. (Auth. Abs.)

84–0876. De La Garza, R.; Johanson, C. E. **The Discriminative Stimulus Properties of Cocaine in the Rhesus Monkey.** *Pharmacology Biochemistry and Behavior* 19(1): 145-148, July 1983.

Nicotine produced inconsistent patterns of cocaine lever responding in three rhesus monkeys trained to discriminate i.m. injections of cocaine (0.25 mg/kg) from saline, under conditions where responding was maintained on one of two levers under a 30 min fixed-ratio schedule of food delivery. The ability of other doses of cocaine and other compounds to substitute for the training dose of cocaine in controlling

cocaine-appropriate responding was assessed. Cocaine (0.008 to 0.5 mg/kg), *d*-amphetamine (0.03 to 0.25 mg/kg) and *l*-cathinone (0.03 to 0.5 mg/kg) produced dose-dependent increases in the percent of cocaine-appropriate responding in test sessions, suggesting that they share discriminative stimulus properties. When nicotine (0.125 to 16 mg/kg) and procaine (0.06 to 8.0 mg/kg) were tested, some of the monkeys responded 90 percent or more on the cocaine-appropriate lever; in other cases there was a trend toward generalization at lower doses. When higher doses were administered, responding was suppressed. Data suggest that the two compounds have discriminative stimulus properties somewhat different from cocaine. Responses to pentobarbital (2 to 16 mg/kg) occurred on the saline-appropriate lever, indicating that this drug does not share the discriminative stimulus properties of cocaine. (Auth. Abs. Mod.)

84–0877. Didisheim, J.-C. **Les Repercussions de la Consommation de Tabac sur les Performances Sportives. [The Repercussions of Smoking on Sport Performance.]** *Therapeutische Umschau/Revue Therapeutique* 40(2): 117-120, February 1983, French.

Among men 30 to 50 years old with high aerobic power, capable of good results in sports requiring prolonged muscular exertion such as running, cycling, or cross-country skiing, only 5 percent are smokers, with none smoking more than 20 cigarettes/day. However, among participants in sports such as tennis or football, which are considered more as recreation than as a means of developing endurance, 35 percent are smokers, of whom 3 percent smoke more than 20 cigarettes/day. It is noted that among men aged 30 to 40 years, only 14 percent smoked (25 percent >20 cigarettes/day), while 35 percent of those aged 40 to 50 years smoked (3 percent >20 cigarettes/day). The physiological mechanisms by which smoking disrupts sports performance are discussed.

84–0878. Eiser, J. R. **Behavioural Medicine: What Kind of Medicine? What Kind of Behaviour?** *Journal of the Royal Society of Medicine* 76(8): 629-632, August 1983.

Practitioners of behavioral medicine are urged to develop their understanding of (1) the social, economic, and political factors that create and legitimize an environment in which smoking is widespread; and (2) the personal and interpersonal factors that predict varying individual responses to environmental elements. A behavioral aspect is imparted to the etiology of the major fatal diseases of the modern world, coronary heart disease and cancer. While, as with other drugs, there is a severe problem of relapse, various studies show that (1) individual psychological treatment for smoking can be beneficial; (2) smokers who seek treatment constitute only a small unrepresentative sample of the smoking population as a whole; (3) most smokers who quit do so without professional help; and (4) the degree to which smokers label themselves as addicted may correspond with their difficulty in quitting. It is proposed that behavioral medicine create an intellectual environment in which the theories and methods of different disciplines may be brought together to define the social and interpersonal implications of most health behaviors.

84–0879. Eysenck, H. J. **A Note on "Smoking, Personality and Reasons for Smoking".** *Psychological Medicine* 13(2): 447-448, May 1983.

While response to a previously published paper on smoking and personality indicates that findings significantly correlating psychoticism (but not extraversion) with smoking are atypical of work with the Eysenck Personality Questionnaire (EPQ), a hypothesis to explain psychoticism as a smoking initiation factor is suggested. Original work on the importance of extraversion as a factor was conducted around 1960, when smoking was socially acceptable and therefore reinforcing to socially oriented extroverts. However, since smoking is currently socially disapproved, extroverts might avoid smoking and thus reduce the expected degree of correlation. Social disapproval might actually attract high psychoticism scorers, since much of their conduct is antisocial, aggressive, and socially undesirable. For these individuals, social disapproval may constitute positive reinforcement. Hence, an increment over the years in the correlation between smoking and psychoticism may have occurred simultaneously with a declining correlation between smoking and extraversion. However, results of a 1983 study using the EPQ (n=900) showed that smokers scored higher than nonsmokers on the extraversion, neuroticism, and psychoticism scales; nonsmokers had higher lie scores than smokers. It is emphasized that a firm understanding of the relationship between smoking and personality can be achieved only through repeated measurement at regular intervals, using identical test instruments administered to populations chosen according to identical criteria.

84–0880. Gedda, L.; Brenci, G. **Twins Living Apart Test: Progress Report.** *Acta Geneticae Medicae et Gemellologiae* 32(1): 17-22, 1983.

In preliminary results of the pilot test, higher concordance values for alcohol and tobacco use were found for the twins still living together compared with those now living apart, underscoring the influence of environmental factors. However, there was also a considerably high concordance level in the twins living apart, supporting the idea that genetic factors may also play a part. Data are from a new concept in twin research that is based on the study of twins who, though reared together, have subsequently lived apart for a period of at least 5 years. Unlike the study of twins reared apart, the twins living apart test has the advantages of being more realistic and affording easier access to sufficiently large samples. The test was applied to a sample of 92 monozygotic pairs now aged 35-45: 15 pairs still living together and 77 that had lived apart for over 5 years. As a first approach, comparisons were made, in the co-twins of the two subsamples, on the following traits: height, weight, presbyopia, presbyacusia, alcohol consumption, tobacco consumption, blood pressure (systolic and diastolic), dental caries, and hours of sleep. (Auth. Abs. Mod.)

84–0881. Knobf, M.K.T.; Morra, M. E. **Smokers, Former Smokers, and Non-Smokers: A Correlational Study of Nurses in Connecticut.** *Oncology Nursing Forum* 10(4): 40-45, 1983.

A 3 percent random sampling of all Connecticut nurses found that 25.5 percent are current smokers, 39.8 percent ex-smokers, and 34.7 percent have never smoked. Answers to the 39-item questionnaire also revealed that (1) smoking was significantly negatively correlated to basic education, but not to continuing professional education; (2) current smokers have smoked for more years than the ex-smokers; (3) smoking nurses do not identify themselves as health educators as frequently as nonsmoking or ex-smoking nurses; and (4) the highest smoking prevalence occurred in the 1921 to 1940 birth cohort. Reasons cited by subjects who had failed to quit included (in order of frequency) loss of determination, stress, weight gain, hyperactivity, and influence of environments predominated by smokers. Surprisingly, 18 percent of the smokers expressed an interest in leading a stop-smoking program. It is also noted that the nurses who smoked reported a higher cigarette consumption while at work. Recommendations for an antismoking campaign targeted at nurses, based on these findings, are included.

84–0882. Krohn, M. D.; Massey, J. L.; Skinner, W. F.; Lauer, R. M. **Social Bonding Theory and Adolescent Cigarette Smoking: A Longitudinal Analysis.** *Journal of Health and Social Behavior* 24(4): 337-349, December 1983.

Research on the viability of social bonding as an explanation of adolescent cigarette smoking is described. Data are based on a two-wave panel study of 1,405 students in grades 7 through 12. The four interrelated elements of the social bonding theory (attachment, commitment, involvement, and belief) are explained as they relate to deviant behavior in adolescents. Questionnaires, administered a year apart, measured elements of the bonding theory. The results generally support the hypothesis that adolescents' ties to aspects of conventional society are important in constraining deviant behavior. Beliefs and commitment to education were found to have the strongest constraining effect, reemphasizing the crucial role of the educational institution in the constraining of adolescent deviant behavior. However, it was found that there was no significant interactive effect of parental smoking and parental attachment on smoking behavior. Also contrary to prediction, commitment to work did not have a constraining influence. The effects of other variables are reviewed, and the significance of the results for understanding adolescent smoking behavior are discussed.

84–0883. Li, V. C.; Kim, Y. J.; Terry, P. B.; Cuthie, J. C.; Roter, D.; Emmett, E. A.; Harvey, A.; Permutt, S. **Behavioral, Attitudinal, and Physiologic Characteristics of Smoking and Nonsmoking Asbestos-Exposed Shipyard Workers.** *Journal of Occupational Medicine* 25(12): 864-870, December 1983.

Rates of pulmonary functions and chest film abnormalities were significantly lower among ex-smokers and never smokers, in an assessment of shipyard workers (n=3,991) participating in an Asbestos Medical Surveillance Program. Sources of information on smoking habits were: (1) a self-assessment questionnaire on the smoking history and respiratory symptomatology of the 871 current smokers who participated in the smoking study; and (2) chest roentgenograms and pulmonary function test results and medical records for the entire population. The study population included 1,711 current smokers, 988 former smokers, and 1,292 never smokers. The annual "quit rate" for former smokers had increased from less than 1 percent in 1961 to 4.2 percent in 1978. Of the 871 current smokers who participated in the smoking study, 19 percent had resumed smoking after having given up cigarettes for 1 year or longer. Men in the smoking study were reasonably well-informed about the health consequences of smoking. While they perceived themselves to be susceptible to disease, and the disease to be serious, the benefits of quitting were seen as related more to economics and esthetics than to health. When results were age-adjusted, no differences in rate of pulmonary function abnormalities and chest film abnormalities were found between current smokers who voluntarily participated in the smoking study and those who did not. (Auth. Abs. Mod.)

84–0884. Loranskiy, D. N.; Popova, Ye. V.; Khakimova, L. S.; Shevchuk, A. G. **Sostoyanie Problemy Kureniya. [The Smoking Problem.]** *Sovetskoye Zdravookhraneniye* 6: 33-38, 1983, Russian.

According to fragmentary data from the early 1960s, 45 percent of Soviet men and 26.3 percent of Soviet women smoked. (The Soviet Ministry of Health began keeping smoking statistics in 1968.) A 1981 poll of 1,687 people in eight Soviet cities indicated that 33 percent of the population were smokers (64.5 percent of men and 11.0 percent of women); the highest rates were found among 20- to 24-year-olds. Of 2,819 schoolchildren (fourth through tenth grade), 18.7 percent smoked (25.9 percent of boys and 11.5 percent of girls). Smoking became common during seventh grade and was highest among the tenth graders (46.2 percent). Gastrointestinal ailments were most common among smokers (36 percent), followed by respiratory ailments (20 percent), and circulatory ailments (11 percent). The chronic illness rate was three times higher among smokers than among nonsmokers. Among persons suffering from gastrointestinal ailments, 26.7 percent occasionally smoked on an empty stomach while 26.6 percent did so regularly. Public attitudes toward smoking and governmental antismoking measures are also discussed.

84–0885. Macdonald, P. T. **The "Dope" on Soaps.** *Journal of Drug Education* 13(4): 359-369, 1983.

Despite the cigarette advertising ban on television, long-term viewers of daytime shows could recall many instances of tobacco use; 75 percent of the 165 soap opera viewers recalled tobacco use, usually rated as a neutral portrayal rather than positive or negative (smoking by extras in bar scenes). Over one-third of the time, however, its use was seen as negative, mainly as a compulsive nervous habit, or in association with health problems. The study examined the context, motives, and consequences of drug use in the daytime serials. In addition to smoking, in-depth analyses of various portrayals of the following drug categories included: alcohol, tranquilizers, amphetamines, marihuana, LSD, cocaine, and heroin. In discussing the potential effects of such portrayals, it is noted that the frequency of viewing, duration of contact, and intensity of the identification with the stars would influence the impact of the message being portrayed. Thus, the casual attitude surrounding cigarette smoking might have latent learning lessons, as would images of women as alcoholics and tranquilizer abusers. The presentation of illicit drugs is especially important due to the audience's probable lack of information about them.

84–0886. Miller, W. R.; Hedrick, K. E.; Taylor, C. A. **Addictive Behaviors and Life Problems Before and After Behavioral Treatment of Problem Drinkers.** *Addictive Behaviors* 8(4): 403-412, 1983.

Smoking cessation was associated with control of drinking, and relapse to smoking was associated with continued alcohol abuse, although initial smoking status was not correlated with alcohol-related treatment among 93 problem drinkers (55 males, 38 females). At baseline, 45 percent were nonsmokers, 4 percent pipe or cigar smokers, and 51 percent cigarette smokers. At 6-month followup, 19 percent of the nonsmokers were smoking, while 22 percent of the smokers had quit and 11 percent had reduced their consumption. Thus, overall rates of alcohol abuse and smoking showed little change. At baseline, women were more likely than men to be unmarried, married to a problem drinking spouse, or taking psychoactive prescription medication; but less likely to be smoking, using illicit drugs, or gambling. For both men and women, negative emotional states and family discord headed the list of current life problems. Although intervention focused primarily on drinking behavior, the majority of other life problems also showed improvement at all followup points. Remission of life problems was associated with decreased alcohol consumption. Body weight tended to increase slightly after treatment but typically returned to or below baseline by later followups. (Auth. Abs. Mod.)

84–0887. Morra, M. E.; Knobf, M.K.T. **Comparison of Nurses' Smoking Habits: The 1975 DHEW Survey and Connecticut Nurses, 1981.** *Public Health Reports* 98(6): 553-557, November-December 1983.

Smoking appears to be decreasing among nurses. A 1981 study of a 3 percent sample of Connecticut nurses (n=545) showed that only 25.5 percent smoked, compared with 38.9 percent in a 1975 nationwide survey. Only 22 percent of the subjects surveyed in 1975 were ex-smokers, compared with 40 percent of the Connecticut nurses. However, 28 percent of the nurses who smoked reported consumption of 25 or more cigarettes/day, compared with 16 percent in the 1975 survey. Also, fewer of the smokers in the 1981 survey agreed that nurses should set a good example by not smoking and that most smokers could quit if they wanted to. No change was found between the 1975 and 1981 surveys concerning attitudes toward smoking cessation in response to physician advice, or the role of the nurse in assisting others to stop smoking.

84–0888. Moskowitz, J. M. **Preventing Adolescent Substance Abuse Through Drug Education.** In: *Preventing Adolescent Drug Abuse: Intervention Strategies.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph No. 47, DHHS Publication No. (ADM) 83-1280, 1983, pp. 233-249.

A literature review presents evidence for the efficacy of antismoking education in reducing cigarette use. The successful programs used cognitive-inoculation to reinforce prevailing antismoking attitudes, and included discussions of the influences that promote smoking, which served to correct students' misconceptions about cigarettes and teach them that most peers and adults share their antismoking attitudes. Thus, the current social climate toward nonsmoking as normative may be an essential ingredient for the success of smoking prevention programss. Also, the programs acted on attitudes at the peer group level; students were early adolescents, and all students participated. Assertiveness training and trained peers as leaders are also common, but not crucial, features of successful programs. In contrast, drug abuse by adolescents does not appear to be amenable to drug education, and current theoretical approaches have little empirical basis. The assumption that adolescents use illicit drugs because of deficient values, inept decisionmaking, or inadequate social skills is unfounded, with little evidence that such deficiencies can be remedied through education.

84–0889. Mueller, R. **Soziokulturelle Determinanten des Rauchverhaltens und Perspektiven der Primarpravention. [Sociocultural Factors of Smoking Behavior and Perspectives of Primary Prevention.]** *Therapeutische Umschau* 40(2): 96-101, February 1983, German.

The significance attributed to smoking by an individual is believed to be preshaped by the cultural environment. An infinite number of situations in which smoking becomes a socially acceptable behavior is an important characteristic of our culture. Social sanctions for smoking are transferred by parents, brothers and sisters, peers, and teachers to children and adolescents. The symbolic sharing in, and simultaneous opposition to, adult values, and the tendency of smoking to help define an adolescent's self-concept, are decisive factors in smoking initiation. Social class, social alienation, occupational stress, peer pressure, and especially the smoking behavior of a partner, are the most important factors contributing to maintenance. Cognitive variables, such as the physical and psychic discomfort experienced in connection with smoking, and support from the social environment contribute to the decision to stop smoking. Smoking is considered not only an expression of self-image and of the adolescent's solidarity within a group, but also a sociocultural ritual. Hence, a primary prevention approach that denies the social logic of smoking is simplistic and inadequate.

84–0890. Newman, I. M. **Talking With Patients About Smoking.** *New Zealand Medical Journal* 96(743, Part 2): 934-937, November 9, 1983.

An algorithm for enhancing physicians' effectiveness in patient smoking cessation counseling is described. Three levels of awareness of the health risks of smoking exist: (1) awareness of seriousness, exemplified by the statement that "the government has determined that cigarette smoking is dangerous to health"; (2) acceptance of seriousness, which attributes the danger to the product (almost 50 percent of smokers surveyed in 1975 were not fully convinced that cigarette smoking is dangerous to health); and (3) personalized acceptance of seriousness. Only 21 percent agreed that "my smoking will have a harmful effect on my health." A health belief model posits that the likelihood of taking a preventive health action (e.g., quitting smoking) depends on perceptions of the seriousness of the condition, personal susceptibility, benefits of quitting, and barriers to quitting. To take advantage of current knowledge of human behavior, a decision tree kept on the physician's desk to help structure antismoking consultations, distribution of pamphlets, and a patient record of such contacts are suggested to help facilitate physicians' success in helping patients quit.

84–0891. O'Farrell, T. J.; Connors, G. J.; Upper, D. **Addictive Behaviors Among Hospitalized Psychiatric Patients.** *Addictive Behaviors* 8(4): 329-333, 1983.

Extensive addictive behavior problems were found in a survey conducted to assess the extent of smoking, alcohol abuse, drug abuse, and obesity among 305 patients hospitalized on other than substance abuse treatment wards of a large psychiatric hospital. Nearly 90 percent had at least one of the problems, and 82.9 percent (253) were cigarette smokers. Compared with nonsmoking patients, smokers were younger, differed in primary diagnosis, and were judged to have greater self-injurious behavior potential. It was noted that only a few patients were receiving any treatment for addictive problems that was likely to be effective. Only 16 percent of the smokers were receiving antismoking treatment, which consisted of restriction of access to cigarettes by the staff. Smokers under treatment were significantly older than the nontreated smokers, and more showed a greater degree of organic brain damage. Thus, prevalence rates for smoking and alcohol abuse substantially exceeded, and for obesity were equivalent to, the prevalence of these behaviors in the general population. Patients receiving treatment were generally cases in which the addictive problem caused a serious immediate danger to the patient or others on the ward or seriously disrupted the ward routine. (Auth. Abs. Mod.)

84–0892. O'Neill, H. K.; Glasgow, R. E.; McCaul, K. D. **Component Analysis in Smoking Prevention Research: Effects of Social Consequences Information.** *Addictive Behaviors* 8(4): 419-423, 1983.

The effects of information about the negative social consequences of cigarette smoking on adolescents' beliefs and intentions to smoke were evaluated. In contrast to the large-scale, multiple component interventions most commonly conducted, this study compared two brief videotaped messages that contained either social consequences or control information: 54 seventh graders enrolled in health education classes participated. Measures collected immediately after the intervention and at 1-month followup indicated that the social consequences information successfully altered subjects' smoking-related beliefs. The social consequences information also affected intentions to smoke in the future, but only for subjects who had previously experimented with smoking. Female smokers' intentions to continue smoking were reduced, but the intervention had the opposite effect on male smokers. The discussion addresses the importance of examining the effects of prevention program components on different subsamples of adolescents in small-scale process evaluations. (Auth. Abs.)

84–0893. Pederson, L. L.; Baskerville, J. C.; Lefcoe, N. M. **Prevalence of and Factors Related to Cigarette Smoking Among Students in Grade 6.** *Journal of Drug Education* 13(4): 305-312, 1983.

Data from 1,428 children, who were in sixth grade in 1975 or 1978, showed that there was no discernible change in prevalence of smoking in this age group over a 3-year period. After controlling for possible confounding variables (curriculum exposure, repetition of a grade, desire to finish high school, attitude toward smoking, knowledge of health effects of smoking, parental and peer smoking, age, and SES), it was determined that neither year of testing nor sex was significantly related to smoking. Peer smoking and attitude toward smoking showed the strongest relationship with cigarette involvement in both surveys. Slightly more females were regular smokers in 1978 (2.9 percent compared with 2.7 percent in 1975) and fewer males (3.0 percent in 1978, 4.2 percent in 1975). The percentage of nonsmokers was also a little higher for both sexes in 1978. Results suggest that the most effective program for prevention of smoking may be found in peer pressure. If educational efforts can develop attitudes that make smoking socially unacceptable, an actual decrease in the proportion of smokers might occur.

84–0894. Prochaska, J. O. **Self-Changers Versus Therapy Changers Versus Schachter.** *American Psychologist* 38(7): 853-854, July 1983.

The conclusions of a study that questioned the efficacy of psychotherapy in treatment of smoking and other addictive disorders (*American Psychologist* 37(4): 436-444, April 1982) are criticized, stating that while addictive disorders are difficult to treat, no evidence indicates that they are impossible. Most of the 49 study subjects who quit smoking did so without therapy. The term "psychiatrogenics" is deemed inappropriate to describe the well-documented deterioration effect, wherein 10 to 15 percent of patients change for the worse under treatment. It is suggested that the conclusions were based on too small a sample, and that the majority of documented comparisons have shown that therapy outperforms absence of therapy. See also 82-0843 and 83–0620.

84–0895. Radovanovic, Z.; Eric, L.; Dimitrijevic, L.; Jamborcic, V. **Cigarette Smoking Among First-Year Medical Students in Yugoslavia and Their Academic Success.** *Journal of American College Health* 31(6): 253-255, June 1983.

Survey results of smoking habits of 317 first-year medical students (129 men, 38 smokers; 188 women, 44 smokers) did not reveal any significant association between smoking and indicators of success for either men or women. The investigation was part of a larger study on psychiatric disorders of students. The freshmen were followed up for 2 years after initial interview, and academic success was assessed in terms of regularity of studying and the mean grade. Among men, the proportion with a low mean grade was higher among smokers compared with nonsmokers; the difference among women was negligible. The finding of no significant difference between smokers and nonsmokers apparently contradicts results of a British study of university students; however, it cannot be expected that the same factors are involved in populations with quite different social and cultural characteristics.

84–0896. Rijintjes, A. G.; Dubois, V. E.; Adriaanse, H. P. **Health Beliefs, Health-Related Behaviour and Professional Performance of General Practitioners: Their Role in Health Education.** Proceedings of the Tenth WONCA World Conference on Family Medicine, Singapore, May 20-24, 1983, pp. 203-208.

A 1981 survey of general practitioners in the Netherlands revealed that slightly, but not significantly, more physicians had ever smoked compared with the general population. However, a significantly larger proportion of the physicians were ex-smokers (67 percent vs. 46 percent of general population). In contrast, 99 percent of the physicians drank alcohol regularly compared with 79 percent of the general population. When asked to rate certain priorities for good health, both groups placed exercise first and abstinence from tobacco second, with nutrition in third place. In rating their own health, 96 percent of physicians vs. 82 percent of the others stated their health was "good" or "very good." Work-related stress was mentioned six times more often by physicians as the single most important unhealthy lifestyle factor. It is noted that the general practitioners rated health as less important and expressed considerably more skepticism toward medical science than the general population.

84–0897. Rose, J. E.; Ananda, S.; Jarvik, M. E. **Cigarette Smoking During Anxiety-Provoking and Monotonous Tasks.** *Addictive Behaviors* 8(4): 353-359, 1983.

Cigarette smokers (n=15, aged 21 to 67) were exposed to three conditions within a single session: stagefright anxiety, monotonous concentration, and a relaxation control. One cigarette was lit during the second 10-minute half of each condition, and smoking topography (number of puffs and cumulative volume smoked) was continuously recorded. Subjects smoked significantly more in the two task conditions than during relaxation, supporting the hypothesis that anxiety-provoking and attention-demanding situations elicit smoking. Younger subjects increased smoking more than older subjects during stagefright, and females responded more than males to the concentration task. (Auth. Abs.)

84–0898. Salas, I.; Ramos, E.; Peters, G.; Pesenti, L.; O'Ryan, F.; Nieme, E.; Montalvan, S.; Molina, A.; Martinez, M.; Manriquez, M.; Mackenna, A. **Prevalencia de Tabaquismo en Adolescentes del Tercer Ano de la Ensenanza Media. [Prevalence of Tobacco Smoking in Adolescents in Third Year of Secondary Education.]** *Revista Medica de Chile* 110(12): 1237-1244, December 1982, Spanish.

Results showed a statistically significant association between cigarette smoking and sociability, and a nonsignificant inverse association between prevalence of smoking and academic performance and/or knowledge regarding health effects. A sample (102 men and 210 women) randomly selected from 3,228 third-year secondary public and private school students in Santiago indicated an increasing prevalence of smoking with age (51.5 percent and 67.9 percent for 15- and 18-year-olds, respectively). Overall prevalence was 56.9 and 59 percent for males and females, respectively (Auth. Abs. Mod.)

84–0899. Sapene, M.; Couturier, C.; Guguen, O.; Perrotte, M. C.; Zourbas, J. **Le Tabac a l'Ecole. A Propos de Trois Enquetes Groupant 2716 Eleves. [Smoking in School. Three Surveys of 2716 Students.]** *Concours Medical* 104(6): 757-758, 761-762, 765-767, February 6, 1982, French.

Survey data show that 41 to 46 percent of students (aged 12 to 18) in three secondary schools of Rennes City, France, are regular smokers. Questionnaire surveys, in 1977 and 1979, also found that, among students aged 11 to 20, (1) girls smoked about as much as boys (48 and 42 percent, respectively); (2) about 10 percent of those 11 to 13 years of age were regular smokers; (3) about 30 percent of the 14- to 19-year-olds smoked more than 10 cigarettes/day; (4) the average age of smoking initiation (for both boys and girls) was 13 years; (5) parental smoking apparently was an influential factor, since 65 percent of students who smoked had at least one parent who smoked; (6) 30 to 50 percent of the smokers had considered trying to quit, but only about half could give a reason for doing so; and (7) 35 percent of the nonsmokers compared with only 20 percent of the smokers had found the first French antismoking campaign useful. It is considered significant that 13 percent of the smokers, but only 3.5 percent of nonsmokers, had tried another drug at least once. It is concluded that antismoking campaigns are necessary but must be supplemented with other prevention measures to modify students' smoking behavior.

84–0900. Schneider, N. G.; Jarvik, M. E. **Time Course of Smoking Withdrawal Symptoms as a Function of Nicotine Replacement.** *Psychopharmacology* 82(1/2): 143-144, December 1983.

Healthy smokers (n=32), recruited through a radio news announcement, provided evidence that withdrawal symptoms are alleviated with the use of nicotine gum (2 mg). Subjects (20 receiving nicotine gum and 12 a placebo gum) provided morning, afternoon, and evening data for 1 week following cessation of smoking. Withdrawal symptoms were measured by Schneider's Smoker Complaint Scale, and carbon monoxide levels were used to verify smoking abstinence. Results show significantly fewer withdrawal symptoms among the group using nicotine gum compared with those in the placebo gum group. A significant interaction between time of day and treatment was also observed; placebo subjects reported increased withdrawal in the evenings compared with morning and afternoon scores, in contrast with those reported by the nicotine gum group. (Auth. Abs. Mod.)

84–0901. Schwartz, J. L. **Myths and Realities of Smoking Cessation.** *New York State Journal of Medicine* 83(13): 1355-1357, December 1983.

Physicians overestimate patients' awareness of smoking hazards and underestimate their own ability to help patients quit. Myths regarding smoking cessation that recur repeatedly among health professionals, smokers, and ex-smokers include: (1) counseling patients is too time-consuming; (2) the public is already aware of smoking hazards; (3) people wish to continue smoking; (4) switching to low-nicotine cigarettes reduces risk; (5) 95 percent of those who quit do so on their own; (6) tranquilizers help smokers quit; and (7) certain methods are just right for certain people. Realities are explored regarding compounds (over 4,000) generated by burning cigarettes; smoking-related mortality, morbidity, and costs; smoking cessation methods, efficacy; motivation (primarily health-related) to quit; validation of smoking behavior through blood or urine tests or measurement of expired carbon monoxide; ineffectiveness of the filter system for stopping smoking; need for followup support; and commitment to quitting as an element for success. While many cigarette-related diseases are diagnosed too late for reversal by smoking cessation, physicians are urged to focus on health promotion to control smoking-related premature disease.

84–0902. Severson, H. H.; Ary, D. V. **Sampling Bias Due to Consent Procedures With Adolescents.** *Addictive Behaviors* 8(4): 433-437, 1983.

Consistent relationships were found between students' self-reports on smoking and cigarette-related questionnaire items, and parental consent to participate in a smoking project. Positive consent was solicited from parents of 604 seventh grade students in four middle schools: 358 (59 percent) returned consents and completed a questionnaire under "bogus pipeline" conditions with saliva and air samples. Two weeks later both students with consent and those without were administered a second questionnaire without physiological measures. Comparison between consent and nonconsent students shows significant differences in the smoking of cigarettes and marihuana, but no difference in the use of alcohol. Additional significant differences were found in exposure to smoking models, and level of education of both parents. The bias shown on significant dependent variables may adversely affect the generalizability of results of studies of adolescent drug use that depend upon positive parental consent. (Auth. Abs. Mod.)

84–0903. Shevchuk, A. G.; Tarasova, R. N. **Ob Organizatsii Sanitarnoprosvetitel'noy Raboty v Bor'byes s Kureniyem. [The Organization of Antismoking Education.]** *Zdravookhraneniye Rossiyskoy Federatsii* 5: 27-28, 1983, Russian.

A total of 1,529 inhabitants (813 men and 716 women) aged 14 to 60 years, in the city of Lipetsk (south-central European Russia) were polled on smoking habits and attitudes. Overall, 36.6 percent of the population smoked (54.2 percent of the men and 5.9 percent of the women). Smoking was most common among adult males (66.1 percent) and adolescents in the final year of secondary school (69.2 percent). The majority (64.4 percent) of smokers began smoking between the ages of 7 and 17. A drawing competition was then organized among sixth graders on the theme "Smoking and Health." The best drawings were exhibited in a local movie theater, and were viewed by more than 2,000 people daily. The exhibit was found to be quite effective in drawing the public's attention to the dangers of smoking.

84–0904. Simms, M.; Smith, C. **Teenage Mothers and Smoking.** *Health Education Journal* 42(3): 87-89, 1983.

Among 456 young mothers (each had a baby before age 20) in England and Wales, 59 percent were current smokers, 12 percent were ex-smokers, and only 29 percent had never smoked. Of those who smoked, 14 percent consumed >20 cigarettes/day, and about 72 percent lived in households with other smokers. In all of Great Britain, 32 percent of teenaged women and 40 percent of 20- to 24-year-old women smoke cigarettes. The young mothers who smoked were more likely to come from a lower social class and a larger family, to be unmarried, living alone with their babies, to have financial problems, and less likely to have educational qualifications. Among the 55 ex-smokers, 26 had quit prior to pregnancy, 17 during pregnancy, and 12 after the birth of their child. Reasons cited for quitting included health (62 percent), feeling sick during pregnancy (31 percent), expense (21 percent), and husband's dislike of the habit (10 percent). Reasons cited for not quitting by current smokers included relief of boredom, help in coping, social isolation, and others that reflected a perceived loss of control over their lives. Three-fifths of the women had been encouraged to quit by their family or household, but only one-tenth had been advised to quit by their doctors.

84–0905. Statistical Bulletin. **Alcohol and Other Drug Abuse Among Adolescents.** *SB-Statistical Bulletin of the Metropolitan Insurance Companies* 65(1): 4-13, January-March 1984.

Use of the two major licit (legal) drugs, cigarettes and alcohol, was more widespread than the use of any illicit drugs among 17,700 high school seniors surveyed in 1982. About 70 percent of the questionnaire respondents had tried smoking at some time, and 30 percent had smoked to some extent during the previous month. Results also showed that (1) cigarettes were used daily by 21 percent, while 14 percent reported consuming a half pack or more daily; (2) daily smoking rates were 29 percent in 1976 and 20 percent in 1981; (3) cigarette use was more prevalent in females, and distinctly lower in the West; (4) only 8 percent of the college-bound students smoked >10 cigarettes/day, compared with 21 percent of the non-college-bound students; (5) no differences were noted between the races; and (6) about 15 percent began smoking before 10th grade, while 9 percent started in grades 10 through 12. Similar data are provided for consumption of alcohol, marihuana, inhalants, hallucinogens, cocaine, heroin, other opiates, stimulants, sedatives, and tranquilizers.

84–0906. World Health Organization. **Guidelines for the Conduct of Tobacco Smoking Surveys of the General Population. Report of a WHO Meeting Held in Helsinki, Finland 29 November - 4 December 1982.** World Health Organization, Helsinki, WHO/SMO/83.4, 1983, 36 pp.

The report presents guidelines for standardizing definitions, measurement methods, and statistics concerning smoking behavior and tobacco consumption. While the guidelines, which are partially based on previous survey experiences and standardization proposals, are designed to enhance uniform collecting and reporting of basic information, different questionnaires accommodate a wide range of variables. Scope and aims of smoking surveys are discussed, as are factors in survey design (including target and age groups, survey types, sample size, sampling procedures, interview vs. self-completion) and operational aspects (task forces, interviewers, pretesting, response rate, underreporting and confidentiality). Methodologies are described for (1) recording sociodemographic variables, smoking behavior, attitudes, and beliefs of both adults (determined according to age of adult responsibility in country under study) and young people; and (2) data analysis (processing and presentation). Basic and comprehensive questionnaires for adult and nonadult age groups are provided.

See also, **84–0699, 84–0725**

SMOKING PREVENTION AND INTERVENTION

84–0907. Andrews, R. L.; Hearne, J. T. **Effects of Primary Grades Health Curriculum Project on Student and Parent Smoking Attitudes and Behavior.** *Journal of School Health* 54(1): 18-20, January 1984.

Recent research indicates that school health programs using cognitive and affective strategies are the most successful in producing behavioral change in adolescents and adults, and that these programs may have secondary benefits for family members. Few studies, however, have investigated the effectiveness of such programs in younger children. The Primary Grades Health Curriculum Project investigated the relationship between an experiential, activity-centered educational program and (1) positive health attitudes; (2) experimentation with smoking and alcohol, and anticipated future smoking; and (3) changes in parental smoking behavior. In 1977, 600 kindergarten students in two New York school districts, pretested for entry level knowledge and health attitudes, were assigned to experimental or control groups. By the end of the third grade, data indicated that experimental students possessed significantly more positive health attitudes, were less apt to have experimented with alcohol and smoking, and were less exposed to peers who engaged in drinking compared with controls. Nonsignificant differences were found for anticipated future smoking. A significant number of parents of experimental group students reported changes in smoking habits as a result of the child's participation in the program.

84–0908. Ball, K. **Pakistan. Attempts to Control Damage by Tobacco Smoking.** *Lancet* 2(8364): 1413, December 17, 1983.

The Pakistan Department of Health and the International Union Against Cancer (UICC) organized a 3-day workshop on control of smoking, which recommended (1) banning all forms of tobacco advertising, including sports promotion; (2) prominent health warnings on all current advertisements; (3) funding by tobacco advertisers for one antismoking message for every three cigarette ads; (4) prohibiting tobacco sales to persons under age 18; (5) smoking habit surveys; (6) facilities to determine cigarette tar and nicotine content; and (7) investigating tobacco crop substitution. The decline in tobacco consumption in Britain, the United States, and other industrialized countries has induced the major tobacco companies to promote consumption in the Third World: 60 percent of British American Tobacco's profits are derived from developing countries. The rapid growth of the tobacco industry in Pakistan is reflected in an eightfold increase in tobacco tax payments, and a corresponding increase in the incidence of lung cancer, coronary heart disease, and chronic bronchitis. About 50 percent of all cancers in males are attributed to tobacco consumption.

84–0909. Botvin, G. J. **Prevention of Adolescent Substance Abuse Through the Development of Personal and Social Competence.** In: *Preventing Adolescent Drug Abuse: Intervention Strategies.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph Series No. 47, DHHS Publication No. (ADM) 83-1280, 1983, pp. 115-140.

The initiation of smoking and other substance use typically begins during adolescence and appears to be the result of the complex interplay of social, personality, cognitive, attitudinal, behavioral, and developmental factors. Traditional smoking, alcohol, and drug education programs have attempted to increase students' knowledge of the risks associated with using these substances. Other programs have attempted to enrich the personal and social development of students through what has been referred to as "affective" education. However, few of these programs have demonstrated any degree of success in terms of the actual prevention of substance use/abuse. Traditional educational approaches to substance abuse prevention appear to be inadequate because they are based on faulty assumptions and are too narrow in focus. The "affective" education approaches appear to have placed too little emphasis on the acquisition of the kind of skills that are likely to increase general personal competence and enable students to cope with the various interpersonal and intrapersonal pressures to begin using tobacco, alcohol, or drugs. Results of research with a broad-spectrum approach to substance abuse prevention are extremely encouraging, but a number of important issues need to be examined further. Large-scale trials will be necessary to determine the effectiveness of this type of prevention strategy with a broad range of adolescents. (Auth. Abs. Mod.)

84–0910. Coleman, M. **The Research Smokescreen. Moving From Academic Debate to Action on Smoking.** *New York State Journal of Medicine* 83(13): 1280-1281, December 1983.

Medical research over the past 30 years has assembled considerable evidence that smoking is the cause of the 20th century lung cancer epidemic and a major etiologic factor in many other diseases. This success has not been translated into improvements in public health because governments have failed to respond. Although health education campaigns have resulted in a decline in smoking rates in Great Britain since 1966 (from 62 to 42 percent in men and from 44 to 32 percent among women), the Government still has no overall strategy to deal with the smoking problem. Parliamentary action has been defeated by those with financial interests in the tobacco industry, and "voluntary agreements" have simply led to indirect (but still effective) rather than direct advertising. The British tobacco industry has even endowed a Health Promotion Research Trust with 11 million pounds; however, studies designed to directly or indirectly examine the use and effects of tobacco products are specifically excluded. While civilized governments have a clear duty to protect the health of the people, many display a corporate addiction to tax revenue from tobacco, and must rediscover the political will to confront the tobacco industry. Physicians are urged to use political clout to defeat smoking just as tuberculosis and cholera were defeated in the past.

84–0911. Ershoff, D. H.; Aaronson, N. K.; Danaher, B. G.; Wasserman, F. W. **Behavioral, Health, and Cost Outcomes of an HMO-Based Prenatal Health Education Program.** *Public Health Reports* 98(6): 536-547, November-December 1983.

A greater percentage of the 57 women participating in an experimental health education program quit smoking during pregnancy compared with the percentage of 72 women receiving standard prenatal care (49.1 percent vs. 37.5 percent). The experimental group (24 smokers of <20 cigarettes/day, 33 of >20 cigarettes/day) received individual nutrition counseling and a home correspondence smoking cessation program. Of those who smoked throughout pregnancy, the experimental group had a greater reduction in daily consumption, and significantly more of the group made diet adjustments (91 vs. 68 percent). Analysis of birth outcome data revealed that infants born to the experimental group had a significantly higher mean birth weight than infants born to the controls and also had fewer low birth weight infants. Hospital treatment cost savings associated with the reduced incidence of low birth weight infants among experimental group women yielded an overall cost—benefit ratio of approximately 2:1. (Auth. Abs. Mod.)

84–0912. Godber, G. E. **A Global View. Health Versus Greed.** *New York State Journal of Medicine* 83(13): 1248-1249, December 1983.

Worldwide developments in the campaign against smoking, since early reports in the 1950s established a link between smoking and lung cancer, are discussed. Recommendations by the World Conferences on Smoking and Health, held every 4 years since 1967 (without promotion by a government or national medical organization) are reviewed, particularly the proposals of the Expert Committees of the World Health Organization that began by urging comprehensive national campaigns and are now supporting international action on economic and agricultural changes. The international approach is needed to reduce the threat to less developed, less healthy, and less affluent countries created by the multinational tobacco conglomerates, whose advances are slowly being contained in the more developed countries. The problem now is not on deciding on what action to take, but how to insure that the action is taken. Governments must make a stand; there are other crops and other industries that might be far more beneficial to the people affected. Voluntary agreements about direct promotion will never be effective as long as indirect methods, such as promotion of cultural and sports events, are left open. The essential elements for control remain the same as set out by the last two conferences: (1) stop commercial promotion of tobacco products; (2) limit smoking in public places; (3) increase the cost of smoking by progressive taxation on tobacco products; and (4) improve health education, especially of children.

84–0913. Harris, J.; Seymour, L. **No Smoking—Still Not a Sign of the Times at Work.** *Occupational Health* 35(7): 308-313, July 1983.

Survey results from 100 randomly selected companies found that the majority (94 percent) did not have any formal smoking policy other than for safety reasons. Of the six that claimed to have formal policies, only one acknowledged that it discouraged smoking to promote the health of employees. However, more than half of the personnel directors interviewed believed that employees' smoking should be of concern to employers. Even companies without formal policies restrict smoking in certain areas, but such bans seem to be due largely to the nature of the area rather than because of a general health policy. A large number of respondents (45 percent) could not see any advantages in adopting a policy against smoking, and 86 percent felt there would be disadvantages. Evidence suggests that companies have a functional attitude toward smoking, and need information on the benefits of a formal smoking policy. Further research is needed into the combination of policies most likely to be effective, and how to promote the acceptance and implementation of such policies.

84–0914. Jeffery, R. W.; Wing, R. R. **Recidivism and Self-Cure of Smoking and Obesity: Data From Population Studies. (Letter).** *American Psychologist* 38(7): 852, July 1983.

Concern is expressed regarding the accuracy of the retrospective interview method used to estimate smoking and obesity rates in a previous paper (*American Psychologist* 37(4): 436-444, April 1982). It is suggested that the success of self-treatment is overestimated when compared with data from larger and more general population bases. When compared with results of the Lipid Research Clinic's (LRC) prevalence study (3,611 males, 3,444 females), the interview sample shows a smaller percentage of females never having smoked and a greater proportion of both males and females reporting cessation. The discrepancy in cessation rates for females is especially striking: Schachter suggests that 30 percent of females are ex-smokers, compared with 17 percent in the LRC data. Difficulty experienced identifying the percentage of people who have never been obese or those who are ex-obese suggests the need for further research in this area. See also 82-0843 and 83–0620.

84–0915. Johnson, C. A.; Solis, J. **Comprehensive Community Programs for Drug Abuse Prevention: Implications of the Community Heart Disease Prevention Programs for Future Research.** In: *Preventing Adolescent Drug Abuse: Intervention Strategies.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph Series No. 47, DHHS Publication No. (ADM) 83-1280, 1983, pp. 76-114.

While community heart disease prevention programs have been targeted at an older population group, they can provide important clues about how research for community drug abuse prevention (including smoking) could be carried out. To date, most programs for youth have been implemented through schools, and have thus been limited by a number of factors that could be overcome by a program that also utilized families, mass media, and community organizations. Findings from four heart disease prevention programs (the Multi-Risk Factor Intervention Trial (MRFIT), the Oslo Study, the Stanford Three Community Study, and the North Karelia Project in Finland) are reviewed, and their implications for community drug abuse prevention research are discussed. It is noted that cigarette smoking was significantly affected by MRFIT, and is a behavior that should also be targeted in any drug abuse program because (1) tobacco is believed to have great addictive potential; (2) smoking is the single behavior with the most adverse consequences for public health; and (3) smoking is closely associated with other drug use. Similar success in reduction of smoking was reported for the Stanford and the Oslo Study. It is concluded that behavioral technologies for prevention, strategies for community organization, and methodologies for community research design and assessment are now sufficiently developed for productive research in community drug abuse prevention to take form.

84–0916. McAlister, A. L. **Social-Psychological Approaches.** In: *Preventing Adolescent Drug Abuse: Intervention Strategies.* U.S. Department of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration, NIDA Research Monograph No. 47, DHHS Publication No. (ADM) 83-1280, 1983, pp. 36-50.

In an overview of the most important social-psychological variables influencing substance abuse behaviors, anticipation of enjoyment was found to be the strongest cross-sectional predictor of smoking. Causal variables are defined as specific (at the societal and individual level) and general. Behavior-specific influences at the level of individual psychology (learned expectations and skills regarding specific behaviors) are discussed and preventive approaches based on modification of such influences are described. General variables (self-image, family relationship, and school relationship) and their importance in modifying specific processes that seemed to be important in adolescent smoking were evaluated, showing that friends' smoking was a much stronger influence on the smoking behavior of students with disturbed family or school relationships compared with those students who were close to their parents and teachers. The importance of self-image in smoking behavior was not supported. In reviewing efforts to intervene in these more general areas, it is noted that even the most sophisticated educational programs for young people may fail in the face of family disturbance or early failure at school. The need for using both specific and general approaches to prevent drug abuse is cited, and large-scale research programs are urged to assess the effects of any experimental prevention programs.

84–0917. McCormick-Pickett, N.; Natanblut, S.; Gelfand, P. W.; Franz, A. G. **Pharmacists' Role in Smoking Prevention and Cessation.** *Progress in Clinical and Biological Research* 130: 99-102, 1983.

Results of a 4-week pilot test of the "Helping Smokers Quit" program, which is especially designed for use by pharmacists, indicate that the smoking cessation effort is a viable program. Pharmacists are able to participate with minimal interference of their schedules, and like the program materials; patients also like the materials. Unlike programs designed for physicians and dentists, the focus is on smoking and drug interactions, an issue of special concern to pharmacists. The kit contains display materials, pharmacist materials, and patient handouts. Sponsors of the program (American Pharmaceutical Association and the National Cancer Institute) are considering nationwide use of the program, based on test results. Some modifications to the kit are suggested, particularly the development of special materials for subsegments of the smoking population (e.g., Spanish-speaking individuals).

84–0918. Perry, C. L.; Telch, M. J.; Killen, J.; Burke, A.; Maccoby, N. **High School Smoking Prevention: The Relative Efficacy of Varied Treatments and Instructors.** *Adolescence* 18(71): 561-566, Fall 1983.

Results of a smoking prevention program in four California high schools indicate that none of the approaches used was significantly better in reducing the onset of smoking or encouraging cessation. The objectives of the program were to (1) reduce smoking among high school adolescents; (2) assess the relative effectiveness of social skills and health effects programs; and (3) compare college students with health educators as program leaders. A traditional health education, fear-arousal approach was used to compare results (control group). Students were matched at pre- and postassessments by anonymous codes. Of the 82 who reported weekly smoking at pretest, 23 percent reported no smoking in the week prior to posttest. The self-reports were validated with low carbon monoxide readings. Due to small sample sizes, none of the differences are statistically significant. It is interesting to note that teachers appeared to be more effective in the classes with the traditional health effects curriculum, whereas college students were more effective in those classes concerned with social pressures. In qualitative and quantitative assessments of program implementation, the only difference noted concerned classroom conduct, which was more problematic in student-taught classes. While the efficacy cannot be determined without longer followup, it is suggested that educational efforts aimed at behavior change may need multiple approaches, models, and messages to be most effective.

84–0919. Puska, P.; Koskela, K. **Community-Based Strategies to Fight Smoking. Experiences From the North Karelia Project in Finland.** *New York State Journal of Medicine* 83(13): 1335-1338, December 1983.

A summary of antismoking activities in the North Karelia project notes that cessation was the primary objective; switching to low-yield brands was not encouraged. The intervention involved the local social organization and existing service structure, with most activities conducted by the community. Local newspapers published about 250 articles on smoking, radio stations broadcast nonsmoking messages, and leaflets and other nonsmoking materials were distributed. Civic organizations were asked to promote nonsmoking in their facilities. Seminars were organized for health professionals, who were then asked to give cessation advice to patients. Smoking cessation courses emphasizing practical quitting skills were arranged. Between 1972 and 1982, women showed a net reduction in smoking of 14 percent, while smoking among men decreased 28 percent. Between 1969 and 1979, coronary heart disease mortality declined 24 percent among middle-aged males and 51 percent in middle-aged females, with most of the decrease evident after the initiation of the program. The results in North Karelia County have prompted national antismoking legislation that will address tobacco promotion, smoke yields, labeling requirements, and other issues.

84–0920. Rosenberg, J. **The AMA Tackles Smoking. "A Strong Stand".** *New York State Journal of Medicine* 83(13): 1363-1365, December 1983.

A review of American Medical Association (AMA) actions on smoking, gleaned from the *Journal of the American Medical Association* and the *American Medical News*, leaves no doubt regarding increasing commitment to reduction of smoking. Members are urged to play a major role through personal example and advice regarding smoking-related health hazards.

Official policy states that smoking is to be discouraged through public pronouncements, educational programs, and a strong antismoking stand by every available means. Research on tobacco and health, as well as the tobacco companies' contributions and relationship with the AMA, are traced from 1963 through 1982.

84–0921. World Health Organization. **Smoking Control Strategies in Developing Countries.** World Health Organization, Technical Report Series 695, Geneva, 1983, 94 pp.

The problem of controlling smoking in developing countries is addressed in a World Health Organization (WHO) Expert Committee report, which notes that developing countries have a unique opportunity to control smoking, the most avoidable of modern epidemics. The report discusses the epidemiological and economic aspects of control; the objectives, structure, data collection, public education and information, restrictive measures and legislation, and smoking cessation as means toward control; research; international activities, including the role of WHO; and recommendations addressed specifically to the problem of smoking in developing countries. Among the recommendations are: (1) provide smoking control activities; (2) coordinate governmental programs; (3) establish a central control agency; (4) conduct special education programs for children; (5) organize community leaders; (6) ban all advertisements and restrict sales, especially to minors; (7) control cultivation of tobacco, and reduce its economic impact by introducing alternative crops; (8) integrate actions into the national health care system; (9) encourage research; and (10) utilize international assistance. Other recommendations, which are interrelated, are directed toward developed countries, WHO, and organizations of the United Nations system. Recommendations of previous committees are appended.

See also, **84–0776**

SMOKING CESSATION METHODS

84–0922. Coates, T. J.; Li, V. C. **Does Smoking Cessation Lead to Weight Gain? The Experience of Asbestos-Exposed Shipyard Workers.** *American Journal of Public Health* 73(11): 1303-1304, November 1983.

The relationship between smoking cessation and weight change in 373 asbestos-exposed workers who had participated in a stop smoking program was examined. Those who quit smoking for 1 year and those who quit for shorter periods gained significantly more weight than those who continued to smoke. Mean weight gain was 5.15 lb for those who quit at 3 months and were abstinent at 12 months, 3.76 lb for those who were smokers at 3 months but abstinent at 12 months, 2.76 lb for those who quit at 3 months but were smokers at 12 months, and 0.35 lb for those who continued to smoke. Those with initially poorer health and those who quit to avoid illness gained less weight. (Auth. Abs. Mod.)

84–0923. Deaton, J. E. **Effectiveness of Isolation in the Reduction of Cigarette Smoking.** *Military Medicine* 148: 750-753, September 1983.

Using a restricted environmental stimulation technique (REST), four subjects reduced smoking by 68 percent compared with baseline frequency. The patients were all males (average age 32) and had smoked an average of 35 cigarettes/day for 16 years. All were exposed to a series of taped messages dealing with various techniques designed to reduce smoking, and spent 24 hours in a dark, sound-reducing chamber. Patients did not appear to experience any undue stress; in fact, results from the Health Scale 6 months after REST treatment showed that patients seemed to experience less tension, anxiety, and headaches over time. In addition, patients indicated an increased ability to fall asleep. In terms of expressed attitudinal changes associated with smoking behavior, results indicated a significant change in orientation toward smoking. Patients stated that they were more likely to maintain greater control over cigarette use and were not as likely to feel that they must smoke to cope with negative emotions. The implications of these findings suggest that the REST program is effective in decreasing previous smoking patterns. (Auth. Abs. Mod.)

84–0924. Erickson, L. M.; Tiffany, S. T.; Martin, E. M.; Baker, T. B. **Aversive Smoking Therapies: A Conditioning Analysis of Therapeutic Effectiveness.** *Behaviour Research and Therapy* 21(6): 595-611, 1983.

Rapid smoking (RS) and a less aversive rapid-puffing (RP) treatment were compared on ability to enhance the impact of a behavioral-counseling (BC) procedure on psychophysiological, attitudinal, and behavioral responses to cigarettes. Subjects (10 women and 6 men; mean age 31.1, mean years of smoking 12.4) were randomly assigned to one of the three groups. Data reveal that both aversive smoking treatments resulted in less smoking once treatment began, reduced smoking in posttreatment taste tests, and superior followup performance at 6 months and 1 year. At 1 year, RS smokers achieved the highest number of days abstinent of any group. Regression analyses showed that confidence ratings of remaining abstinent were highly predictive of followup status, and that ratings were related to number of cigarettes smoked during treatment and amount of smoking during posttreatment taste tests. Despite some procedural and outcome differences between the RS and RP treatments, both appeared to produce therapeutic effects in a similar manner: a rapid cessation of smoking once treatment began, and a significant reduction of smoking during taste tests (probably reflecting conditioned aversions to cigarettes). (Auth. Abs. Mod.)

84–0925. Murphy, G. P.; Sciandra, R. **Helping Patients Withdraw From Smoking.** *New York State Journal of Medicine* 83(13): 1353-1354, December 1983.

While all physicians agree that eliminating cigarette smoking is an essential aspect of health promotion, most underestimate their ability to help patients quit. Of 433 primary care physicians surveyed in Massachusetts, 90 percent routinely asked patients about smoking, 58 percent were prepared to counsel patients, but only 3 percent were very confident regarding counseling success. However, it is indicated that over one-half million smokers in Great Britain would quit each year if general practitioners simply advised them not to smoke. Smokers considered especially receptive to counseling include pregnant women, severely ill (especially myocardial infarction) patients, and those visiting a physician's office or other health care setting. The physician should not consider any patient who continues to smoke as compliant, even when the patient adheres to other recommendations. While there is no single best way to counsel patients, physicians are advised to clearly communicate strong feelings against smoking, encourage patients without relying heavily on fear or guilt, agree on a target date, offer continuing encouragement and advice, prepare patients for side effects, and provide aid in identifying situational smoking cues and alternate activities. Suggested smoking cessation materials are listed.

84–0926. Wagner, T. J.; Hindi-Alexander, M.; Horwitz, M. B. **A One-Year Follow-Up Study of the Damon Group Hypnosis Smoking Cessation Program.** *Journal of the Oklahoma State Medical Association* 76(12): 414-417, December 1983.

Evaluation of the Damon group hypnosis smoking cessation program revealed that of 783 subjects who participated, 113 had not smoked in the year following treatment. Aggressive data collection methods yielded a 1-year followup response rate of 79 percent, 48 percent from questionnaires and 31 percent from a telephone survey. In addition to the 14 percent who were ex-smokers since hypnotherapy, 2 percent smoked regularly after therapy but quit during the following year, 3 percent smoked occasionally, and 59 percent remained regular smokers. The telephone survey respondents were significantly less likely to be abstinent at followup than the questionnaire respondents. Nonrespondents were counted as failures. It is noted that the self-report methodology of this study is its major limitation.

TOBACCO PRODUCT ADDITIVES

84–0927. Boden, R. M. **Organoleptic Use of Prins Reaction Products of Diisoamylene Derivatives.** United States Patent No. 4,359,412, November 16, 1982, 52 pp.

A process is described that provides Prins reaction products for imparting oriental, fruitwood-like, rum-like, and Turkish tobacco-like aroma and taste nuances to smoking tobacco and substitute smoking tobaccos prior to and on smoking, in both the mainstream and sidestream smoke. The Prins reaction is carried out on diisoamylene, with formaldehyde or a formaldehyde precursor such as formalin or paraformaldehyde, in the presence of or in the absence of an acyl anhydride and in the presence of an acid catalyst; either a Lewis acid such as borontrifluoride etherate or stannic chloride or the like, or a protonic acid such as sulfuric acid or phosphoric acid. Similar compositions are described in 84-0931. (Auth. Abs. Mod.)

84–0928. Boden, R. M. **1-Ethoxy-1-Ethanol Acetate-Acetaldehyde Mixtures for Augmenting or Enhancing the Aroma of Detergents.** United States Patent No. 4,387,036, June 7, 1983, 19 pp.

An organoleptically improved smoking product, containing as an active ingredient the mixtures of 1-ethoxy-1-ethanol acetate and acetaldehyde described, has desirable sweet, fruity, winey, fresh berry-like notes. The additives may be readily varied and controlled to produce the desired uniform flavoring characteristics. Satisfactory results are obtained if the proportion by weight of 1-ethoxy-1-ethanol acetate to smoking tobacco material is between 50 ppm and 1,500 ppm. Any convenient method may be employed for incorporating the mixture into the tobacco product (or filter), and only a portion of the tobacco or substitute needs to be treated. The mixtures may also be used to add flavor and aroma to foodstuffs, chewing gums, toothpastes, medicinal products, and perfumes.

84–0929. Boden, R. M. **Aliphatic Branched Olefin Dioxolanes.** United States Patent No. 4,374,998, February 22, 1983, 52 pp.

The synthesis and uses of various branched chain olefin dioxolanes, dithiolanes, and oxathiolanes as a smoking and chewing tobacco flavorant are presented. The compounds described enhance the cedar, amber-like, woody, sweet, and vetiver-like aroma and taste nuances of black and cigar tobacco

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smoke. Satisfactory results are obtained with 50 to 15,000 ppm, or 0.015 to 1.5 percent by weight. The dithiolanes, dioxolanes, and oxathiolane compounds described are derived from diisoamylene, and may also be used with foodstuffs, chewing gums, medicinal products, perfumes, and perfumed articles. Similar compounds are described in 84-0930.

84–0930. Boden, R. M. **Aliphatic Branched Olefin Dioxolanes, Dithiolanes, and Oxathiolanes and Uses Thereof in Augmenting or Enhancing the Aroma and/or Taste of Consumable Materials.** United States Patent No. 4,381,243, April 26, 1983, 55 pp.

The dithiolane, dioxolane, and oxathiolane compounds described augment or enhance the sweet, woody, vetiver-like, and cedar-like aroma and taste nuances of black tobacco and cigar tobacco both prior to and on smoking in both the mainstream and sidestream smoke. The compounds also augment and enhance the nutty, earthy, woody-balsamic, walnut-kernel like and walnut skin flavor and aroma nuances in chewing tobaccos, foodstuffs, chewing gums, medicinal products, and toothpastes. The desired compound may be added to the chewing tobacco, smoking tobacco, or to part of the smoking tobacco article such as a wrapper or filter. Any convenient method for incorporating the compounds may be used. Satisfactory results are obtained if the proportion by weight of the sum total of the described compounds to smoking tobacco material is between 50 ppm and 1,500 ppm. The compounds may be mixed with other additives so that the combined odors produce a pleasant and desired fragrance. Similar compounds are described in 84-0929.

84–0931. Boden, R. M. **Organoleptic Use of Prins Reaction Products of Diisoamylene, Derivatives Thereof, Organoleptic Uses Thereof and Processes for Preparing Same.** United States Patent No. 4,381,242, April 26, 1983, 44 pp.

A process is described that produces Prins reaction products of diisoamylene useful in imparting oriental, fruity, rum-like, and Turkish tobacco-like aroma and taste nuances prior to and on smoking in mainstream and sidestream smoke. The materials have the further advantage of being stable over time. They may also be used to augment and enhance various flavor and aroma characteristics of chewing tobaccos, foodstuffs, chewing gums, medicinal products, toothpastes, perfumes, and perfumed articles. For smoking tobaccos, satisfactory results are obtained if the proportion by weight of the sum total of one or more of the Prins reaction product derivatives to smoking material is between 250 ppm and 1,500 ppm. Any convenient method for incorporating the derivatives in the tobacco product may be employed, and the derivatives may be used alone or with other flavoring additives. Similar compositions are described in 84-0927.

84–0932. Cline, W. K. **Wrapper for Smoking Articles and Method.** United States Patent No. 4,420,002, December 13, 1983, 6 pp.

A wrapper for cigarettes, cigars, and similar smoking articles is described. The wrapper consists of a cellulosic sheet containing a filler of fine grain magnesium hydroxide (having an average particle size < 10 mm), and unreactive magnesium oxide. The wrapper significantly reduces visible sidestream smoke during static burning and can be used in place of conventional wrappers or as an inner wrapper for the tobacco column. A method for using the wrapping material to produce cigarettes with reduced yields of sidestream tar and visible sidestream smoke, while giving a light colored solid ash that tends to hold on and not flake off, is detailed.

84–0933. Haut, S. A.; Comes, R. A. **Process for Synthesis of N-(Hydrocarbyl)Substituted-p-Menthane-3-Carboxamide.** United States Patent No. 4,366,317, December 28, 1982, 7 pp.

An improved process for producing N-(hydrocarbyl)substituted-p-menthane-3-carboxamide, an odorless and nonvolatile compound that can be used as a physiologically active coolant for cigarettes, is described. The process comprises (1) reacting 3-p-menthyl halide with magnesium in an anhydrous solvent medium to form 3-p-menthylmagnesium halide; and (2) reacting the 3-p-menthylmagnesium halide with hydrocarbyl isocyanate at a temperature below about -20°C to form N-hydrocarbyl-p-menthane-3-carboxamide.

84–0934. Klemarczyk, P. T.; Sanders, J. M.; Vock, M. H.; Vinals, J. F.; Schmitt, F. L.; Granda, E. J. **Carbomethoxy Methyl Norbornanes.** United States Patent No. 4,350,823, September 21, 1982, 15 pp.

A process is described for producing carbomethoxy methyl norbornanes that are useful for augmenting or enhancing the aroma or taste of consumable materials such as chewing tobaccos, chewing gums, foodstuffs, medicinal products, toothpastes, perfumes, and other perfumed articles. The compounds may be used as is and purified by distillation and preparative chromatography; and/or may be further reacted with a reducing agent such as hydrogen to form additional compounds.

84–0935. Light, K. K.; McGhie, J. A.; Fujioka, F.; Yoshida, T. **Use of Tricyclic Alcohols, Ethers and Esters for Augmenting or Enhancing the Aroma or Taste of Foodstuffs.** United States Patent No. 4,357,360, November 2, 1982, 62 pp.

A process is described for producing novel tricyclic alcohols, ethers, and esters that provide oriental-like, herbaceous, minty, earthy, woody, natural tobacco-like, and cedarwood-like aroma and taste nuances to smoking and chewing tobaccos. The compounds can also be used for perfumes, detergents, and foodstuffs. The specific flavoring adjuvants selected will depend on the desired physical form of the ultimate product, and should be capable of providing an environment in which the tricyclic alcohols, ethers, and/or esters can be dispersed or admixed to provide a homogenous medium.

84–0936. Mookherjee, B. D.; Trenkle, R. W.; Wolff, R. K.; Boden, R. M.; Yoshida, T. **Use of Methyl Substituted Pinyl Oxopentenes, for Augmenting, Enhancing of Modifying the Aroma or Taste of Smoking Tobacco and Smoking Tobacco Articles.** United States Patent No. 4,428,387, January 31, 1984, 68 pp.

The processes for preparation and uses of methyl-substituted pinyl oxopentenes as tobacco flavorants are described. When added to tobacco, the mixture imparts various Virginia-type, oriental-type, and Turkish-type tobacco notes, and enhances the natural, sweet, woody, and/or oriental notes in the leaf. The recommended proportion by weight is 200 ppm to 2,000 ppm, or 2,500 ppm to 15,000 ppm. The oxopentenes described may be prepared by reacting alpha or beta pinene according to a standard oxo reaction. The resulting aldehydes may be used as is, or further reacted with methy ethyl ketone under standard "aldol" condensation reaction conditions. The resulting reaction mixture is then distilled and, if desired, reduced with lithium aluminum hydride or similar reducing agents.

84–0937. Ohloff, G.; Giersch, W. K. **Unsaturated Alicyclic Compounds, Their Use as Perfuming and Flavoring Ingredients.** United States Patent No. 4,372,881, February 8, 1983, 4 pp.

Novel unsaturated alicyclic derivatives of pent-4-en-1-one (having a methyl radical bound to the 5 or 6 carbon atom of the ring), which are suitable for use as tobacco flavorants, are described. The compounds are characterized by a strong fruity note reminiscent of pineapple, accompanied by a resinous and green note reminiscent of the aroma of galbanum oil. The compounds are new chemical entities, formed by reacting a carbonyl compound with an allyl halide in the presence of a strong base, followed by hydrolysis of the reaction products.

84–0938. Pittet, A. O.; Muralidhara, R.; Vock, M. H.; Luccarelli, D., Jr.; Schmitt, F. L.; Vinals, J. F.; Kiwala, J. **Flavoring Foodstuffs and Chewing Gum with 4,4A,5,6-Tetrahydro-7-methyl-2-(3H)-naphthalenone.** United States Patent No. 4,385,073, May 24, 1983, 33 pp.

The 4,4A,5,6-tetrahydro-7-methyl-2-(3H)-naphthalenone described, having a purity of 85 percent or greater, and in either liquid or crystalline form, is useful as a tobacco flavorant. The compound and the formulations presented impart various sweet, lactonic, coconut-like, creamy, coumarin-like, waxy, heliotropin-like, fruity, juicy, rum-like, sugary, and woody aroma characteristics to tobacco products, and additional caramelic taste characteristics to tobacco smoke. Optimal concentration of the flavorant is between 100 ppm and 10,000 ppm (0.01 to 1.0 percent by weight). The compound can also be used to flavor foodstuffs, chewing gums, medicinal products, toothpastes, perfumes, and perfumed articles.

84–0939. Schreck, R. P.; Light, K. K.; Hall, J. B.; Schmitt, F. L.; Vock, M. H.; Schreiber, W. L.; Vinals, J. F.; Kiwala, J. **Use of Esters of 1-Acetyl Cyclohexanol in Augmenting or Enhancing the Aroma or Taste of Foodstuffs.** United States Patent No. 4,385,072, May 24, 1983, 10 pp.

The 1-acylcycloalkyl alkanoates produced by the described process impart sweet, minty, cooling, spicy, anisic, berry-like, fruity, pineapple-like, woody, green, pepper-like, and dill-like aroma and taste nuances in tobacco smoke. The alkanoates may also be used to augment the aroma or taste of foodstuffs and chewing gums.

84–0940. Schreck, R. P.; Light, K. K.; Hall, J. B.; Schmitt, F. L.; Vock, M. H.; Schreiber, W. L.; Vinals, J. F.; Kiwala, J. **Process for Enhancing the Organoleptic Properties of Perfumed Articles Using Alkyl Esters of 1-Alkanolyl Cycloalkanols.** United States Patent No. 4,359,395, November 16, 1982, 25 pp.

The manufacture and uses of 1-acylcycloalkyl alkanoates for tobacco flavor enhancement are reported. The compositions described impart sweet, minty, cooling, spicy, anisic, berry-like, fruity, pineapple-like, woody, green, pepper-like, and dill-like aroma notes to smoking tobacco. A quantity between 250 ppm and 1,500 ppm (0.025 to 0.15 percent by weight) is recommended, but up to 15,000 ppm (1.50 percent by weight) is acceptable.

84–0941. Sprecker, M. A.; Hall, J. B. **Substituted Tricyclodecane Derivatives, Processes for Producing Same and Organoleptic Uses Thereof.** United States Patent No. 4,358,617, November 9, 1982, 50 pp.

A process is described for preparing substituted tricyclodecane derivatives that provide cocoa-like, sweet, floral, and green aroma profiles for smoking tobacco prior to smoking, and sweet, floral, and aromatic aroma and taste nuances on smoking in both the mainstream and sidestream smoke. The compositions are racemic mixtures rather than individual stereoisomers, and may also be used in perfumes, detergents, fabric softeners, foodstuffs, chewing gums, toothpastes, and medicinal products.

84–0942. Yoshida, T. **Methyl Substituted Oxobicyclo-4,4,0-Decane Derivatives, Process for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,377,714, March 22, 1983, 87 pp.

A process is described for preparing methyl-substituted oxobicyclo-4,4,0-decane derivatives that provide sweet, fruity, berry-like, cooling, woody, and floral notes to smoking tobacco products. Satisfactory results are obtained if the proportion by weight of the derivatives to smoking tobacco material is between 50 ppm and 1,500 ppm. Any convenient method may be used for incorporating the derivatives into the tobacco product, and only a portion of the tobacco needs to be treated, or the derivatives may be incorporated with filter materials, seam paste, packaging materials, and the like. The derivatives can also be used to enhance the aroma or taste of perfumes, perfumed articles, foodstuffs, chewing gums, toothpastes, medicinal products, and chewing tobaccos.

TOBACCO MANUFACTURING AND PROCESSING

84–0943. Berger, R. M. **Tobacco Smoke Filter Contoured to Provide Undiluted Air Flow and Method and Apparatus for Manufacturing Same.** United States Patent No. 4,423,744, January 3, 1984, 10 pp.

An improved tobacco smoke filter is described that serves as a cigarette holder, permitting undiluted air delivery to the smoker while maintaining the taste of the tobacco smoke. The filter is characterized by an undiluted air passage formed between a smoke-impervious plug wrap and an air-impervious tipping wrap by loosely wrapping the tipping wrap about the plug wrap along a section of the filter extending from its smoke discharge end. Loose wrapping is effected by reducing the cross-sectional perimeter of the rod and plug wrap along the aforesaid section while keeping the tip of the cross-sectional perimeter substantially constant throughout its length. A raised lip is provided at the smoke discharging end of the rod and plug wrap to define a cigarette holder in the filter. For circumstances where the cross-section of the filter plug becomes too complex for adherence of the tipping paper, heat shrinkable film can be laminated in the tipping paper so that after the entire cigarette is assembled, it can be subjected to heat and cause the tipping paper to shrink to the appropriate size. In addition, particles of material which impart flavor to the smoke may be embedded into the plug wrap. Descriptions of the apparatus and method for manufacturing the filter are included. (Auth. Abs. Mod.)

84–0944. Bokelman, G. H.; Gooden, D. T., III. **Acid Curing of Tobacco.** United States Patent No. 4,347,859, September 7, 1982, 4 pp.

A process for artificially curing mature green tobacco is described, by which the tobacco is immersed in an acidic medium and incubated at least at room temperature until the desired color develops. Incubation at pH 1.5 to 3.5 at about 50°C for as little as 3 hours may be sufficient to eliminate the green color and green smoke taste and odor of the tobacco. (Auth. Abs.)

84–0945. Cashwell, E.; Grollimund, E. C.; Duley, W. W.; Lilly, A. C., Jr.; Claflin, W. E.; Stultz, E. B.; Martin, P. **Apparatus for Laser Perforation of Transported Articles.** United States Patent No. 4,349,719, September 14, 1982, 9 pp.

An improved method and apparatus for laser perforation of otherwise complete filter cigarettes are described. The apparatus has components for conducting light energy simultaneously to spaced circumferential locations on a cigarette while it is going through an assembly unit. The assembly unit includes a rotative drum that supports the cigarettes for perforation and a synchronized process of drum rotation and laser pulsing. The method can be used without changing the production efficiency of conventional operations.

84–0946. Hayes, E. B. **Cigarette Filters.** *Tabak Journal International* 5: 433-434, October 1983.

The contemporary cigarette filter (actually a smoke modifier) affects the various fractions of smoke in different ways and can be designed for specific requirements. Cellulose acetate filters reduce smoke harshness and selectively retain polar compounds such as phenols and aldehydes, and tar (to a greater extent than nicotine). Pure cellulose (paper) filters exhibit little sensitivity, although alkaline additives will allow nicotine to pass into the smoke stream without changing tar retention. The creation of multiple filters that combine the advantages of various materials and additives made possible great flexibility in smoke filtration capability. Smoke dilution via ventilation and/or porous papers was added to filtration capacity, allowing engineering of smoke yields for specific market requirements and mandating computerized design for optimal results. The effects of filter geometry, problems with pressure drop, use of flavorants, standard units of measure, the components of smoke involved, and a history of filter use are also discussed.

84–0947. Innacelli, F. L. **Tobacco Smoke Filter.** United States Patent No. 4,373,539, February 15, 1983, 9 pp.

The filtration route of tobacco smoke is increased by coiling a length of plastic tube filled with activated charcoal to form a coiled configuration, which is placed between the conventional cellulose filter and the tobacco charge. Thus, the smoke from the burning tobacco charge starts its filtering route much farther away from the cellulose filter and is cooled and filtered of all tars. The coil may be constructed by molding the activated charcoal as a cone, which is then sprayed with a sealant prior to insertion into a tube. Another means of constructing the coiled configuration is to tap a length of lightweight plastic rod with a "corkscrew" type thread through the longitudinal axis of the rod to form an elongated filtration route. A chemically treated wick to remove tars may be used in lieu of activated charcoal when utilizing the plastic tube for the coiled configuration. (Auth. Abs. Mod.)

84–0948. Lorenz, H.-W.; Schumacher, G.; Kirchner, H. **Apparatus for Determining the Effects of Dilution and/or Diffusion on the Gaseous Components of a Gas Flow.** United States Patent No. 4,348,887, September 14, 1982, 6 pp.

An apparatus for determining the effect of dilution and/or diffusion of the gaseous components of a gas flow passing through a gas-permeable test object is claimed. The apparatus comprises a source of calibrating gas, a laminar resistance coupled to the source of calibrating gas, through which the gas flows, and a volumeter coupled to the laminar resistance for measuring the volume of gas flowing through. The gas from the laminar resistance flows through the test object to a device for receiving and for measuring its concentration. A bypass device is provided for directing the flow of gas around the test object. Suction draws gas from the source to the measuring device. (Auth. Abs. Mod.)

84–0949. Mochida, N.; Morifuji, K.; Sakai, T. **Method for Manufacturing Cigarette Filters.** United States Patent No. 4,357,188, November 2, 1982, 7 pp.

A method for manufacturing cigarette filters is described, by which acetate fibrous tows, impregnated with carboxylic acid ester of polyol, are formed into a predetermined shape, uniformly impregnated with water, and irradiated with microwave energy. Water is used during the application of microwave energy to enhance energy absorption and hence heating of the filter. This causes the carboxylic acid ester of polyol to disperse very rapidly and permeate the acetate fiber, rapidly completing the stabilization and solidification of the filter. (Auth. Abs. Mod.)

84–0950. Rosen, L. M. **Cigarette Tube.** United States Patent No. 4,386,616, June 7, 1983, 4 pp.

The design for a tubular safety enclosure, adapted to be fitted about a cigarette while being smoked to minimize fire hazards, is described. The enclosure is provided with two sections, one suitable for engagement with the filter end of the cigarette and the other providing an enclosure for the tobacco portion of the cigarette. The smoker may thus continue to engage his lips with the filter portion of the cigarette in the conventional manner while being provided with a safety enclosure. The apparatus is intended to minimize safety hazards and may be either reusable or disposable, depending on type of manufacture. Preferably, it is made of an inexpensive heat-resistant and fireproof plastic material. (Auth. Abs. Mod.)

84–0951. Schweitzer, R. A.; Tate, J. H. **Smoke Filtering and Cooling Device.** United States Patent No. 4,357,948, November 9, 1982, 6 pp.

A smoke filtering and cooling device is described that is removably mounted within a cylindrical vertical passage in a tobacco water pipe through which smoke is upwardly directed. The device serves to define passageways whereby the smoke is directed through the water for filtering and cooling as it passes through the pipe. It is comprised of a selectively expandable/retractable portion that is engageable with the passage wall, to provide mechanical support, and a fluid-tight seal, so that a quantity of water can be retained in the passage around the device. A plurality of these devices may be arranged one above another to increase efficiency. (Auth. Abs. Mod.)

84–0952. Silberstein, D. A.; McMurtrie, A. **Cigarette Filter.** United States Patent No. 4,424,819, January 10, 1984, 7 pp.

A cigarette filter is described that lowers tar by ventilation while providing filtration of tobacco smoke. The filter includes a porous filter rod circumscribed by a nonporous or air-impermeable wrapper. Ventilating air grooves are embedded in the wrapped filter rod and extend from one end of the rod a preselected distance. The ends of the grooves are recessed a predetermined depth and communicate with radially extending open channels formed in the one end of the filter rod. An air-permeable tipping material surrounds the wrapped filter rod, providing a path for ventilating air flow into the grooves. The air flowing in the grooves is separated from the smoke flowing through the filter rod by the air-impermeable wrapper, so that ventilating air is the only substance flowing in the grooves. (Auth. Abs. Mod.)

84–0953. Tabak Journal International. **BARCLAY Hearings Prompt Investigation Into FTC Testing Methodology. US Cigarette Industry's Response to the FTC Inquiry. (Excerpts).** *Tabak Journal International* 5: 390-391, October 1983.

When the Federal Trade Commission (FTC) decided that Barclay cigarettes yielded higher tar, nicotine, and carbon monoxide than levels registered by testing machines, the accuracy of the machines came under careful consideration. As a result, the FTC approached the six major cigarette manufacturers for thoughts on the matter, and asked whether or not the existing machines should be altered for Actron filters. The specific questions were: (1) should cigarette testing methods be changed for Barclay, (2) are Kool Ultra and Kool Ultra 100s accurately tested by current methods; (3) which proposed testing filter is best for the Actron filter (MK II "Filtrona," Modified Cambridge, or a filter that blocks air vents); (4) should the testing methods for all cigarettes be modified; (5) how quickly and easily and what would be the cost of a testing modification; (6) what would be the spillover effects of testing changes; and (7) should a banded range of tar content be used in place of actual numbers? Responses by the various manufac-

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turers, who did not always agree, are included. (Auth. Abs. Mod.)

84–0954. Terasaki, M. **Cigarette Holder Smoke Filtering Cartridge With Two Stage Smoke Purification Action.** United States Patent No. 4,370,988, February 1, 1983, 10 pp.

The outer shell of a removable filter cartridge for fitting inside a cigarette holder is formed from two coaxially abutting tubes separated by a wall with a hole. A tubular inner lip around the hole extends into the space within the first tube, a trough being defined around this lip. A cup is mounted within the first tube remote and facing away from the wall, intercepting across the first tube except for one or more narrow spaces defined between the cup and the first tube inner wall. The parts of these narrow spaces nearer the wall communicate to the space between the cup and the wall. One or more small apertures are formed through the side wall of the cup and communicate the space within the cup to the narrow spaces. A disk is mounted across the first tube against the side of the cup remote from the wall. Smoke passes through a hole in the disk into the space within the cup, passes through the small apertures to enter the narrow spaces, and impinges against the inner wall of the tube, to which the larger tar particles in the smoke stick. Then the smoke passes through the narrow spaces, sweeping these tar particles along and depositing them in the trough, and passes through the hole in the wall into a filter mounted within the second tube, which purifies the smoke of some of the smaller tar particles still in it. (Auth. Abs.)

84–0955. Utsch, F. V.; Merritt, H. B.; Sykes, L. M. **Expansion of Tobacco.** United States Patent No. 4,366,825, January 4, 1983, 8 pp.

A tobacco expansion process using a tangential separator, high temperatures, and a short entrainment time, is described. The tobacco and a heated gas stream (at least 525°F) pass through an inlet pipe to a tower unit, then on to the tangential separator. Residence time in the tower unit is approximately 0.2 to 2.0 seconds. An increased filling power of 78 cc/10 g of tower exit moisture is achieved.

TOBACCO ECONOMICS

84–0956. Clairmonte, F. F. **The Transnational Tobacco and Alcohol Conglomerates. A World Oligopoly.** *New York State Journal of Medicine* 83(13): 1322-1323, December 1983.

The world of tobacco politics and power is discussed, noting that the health element is a very minor aspect. Tobacco manufacturing in the developed and underdeveloped capitalistic economies is dominated by seven giant transnational conglomerates whose world sales exceeded 56 billion dollars in 1979. The growth of such conglomerates has been made possible not only by the addictive nature of the commodity, but also by the global working relations between finance capital and the industry. The tobacco companies thus can exert enormous pressure and influence the direction of research to eliminate certain kinds of research regarded as inimical to their interests. An increasingly significant commodity over recent years has been alcohol; e.g., Philip Morris (the world's number three tobacco company) has become the world's number two producer of beer and is heavily involved in wines. Other conglomerates have similar holdings. The unifying factor of these firms is that they are oriented toward the global market, more important as consumption in some developed markets (e.g., United States) has shown signs of stagnation. The tobacco industry must be studied within a political and economic framework as well as a social context.

84–0957. Gabriel, L. **Market Trends for Cigars and Cigarillos in the Federal Republic of Germany.** *Tabak Journal International* 5: 382, 384-385, October 1983.

In 1982, 65 companies (94 factories) in West Germany manufactured 1,733.4 million cigars, cigarillos, and cheroots, which represented a drop in production of 13.8 percent compared with 1981. Whereas sales of cigarillos declined by 15.3 percent, sales of cigars weighing >3 grams were practically unchanged. There was also a reduction in imports and exports during 1982. However, sales were more brisk in the first half of 1983, reaching 919 million cigars and cigarillos, an increase of 30 million pieces or 3.4 percent. The cigar industry is intensifying sales efforts, which are aimed at younger consumers and include certifying some specialized tobacco retailers as "cigar experts." (Auth. Abs. Mod.)

84–0958. Gooch, P. **U.S. Cigars: On the Brink of Recovery?** *Tobacco Reporter* 110(11): 36-38, 40, 42, November 1983.

Over 40 million Americans are approaching the cigar-smoking age of 35, representing potential growth for the cigar industry. Over the past 10 years, cigar consumption declined 48 percent; 1981 sales were 3.9 billion compared with 6.9 billion in 1973. However, consumer expenditures increased by 5.5 percent between 1981 and 1982. The cigar market has two distinct segments: premium high-priced cigars aimed at more affluent, experienced cigar smokers; and lower-priced, mass-produced, flavored cigars which appeal to younger smokers, older, less affluent cigar smokers, those who simply enjoy their taste, and consumers making a transition from other tobacco products. It is noted that cigarette smokers are trying small cigars in response to cigarette price increases; the cigars have more tobacco and are perceived as a better value than low-yield cigarettes. Strategies used to capture this new market will emphasize aggressive marketing techniques to change the image of cigars; innovative, cost-efficient manufacturing methods; and fewer new brands.

84–0959. Ledwith, F. **Smoking Policy and the Tobacco Industry: A Multi-National Problem of Economic Development.** *International Journal of Health Education* 2(1): 17-21, March 1983.

The tobacco industry, dominated by seven transnational conglomerates, exercises tremendous economic power, indicated by the 1.8 billion dollars spent on global advertising in 1978 (backed by social research and the necessary marketing infrastructure). Political influence is also involved, including payoffs to plant politicians and removal of uncooperative officials. In addition, the ambivalence of governments that depend on and encourage tobacco production while giving token support to smoking-related health issues actually encourages tobacco industry exploitation of the Third World. However, despite its ability to act globally, the tobacco industry is not all-powerful. It has two major areas of weakness: the issue of smoking's social acceptability, and the issue of Third World multinational exploitation. Since smoking is closely linked with economic development, global smoking control will require a more equitable sharing of the world's resources. Voluntary controls by the industry are considered placebos, ineffective and often counterproductive. If smoking is to be controlled, governments of Third World countries must be persuaded to reduce their reliance on tobacco by offering alternative means of economic development.

84–0960. Lokschin, F. L.; Barros, F. C. **Smoking or Health: The Brazilian Option.** *New York State Journal of Medicine* 83(13): 1314-1316, December 1983.

In Brazil, cigarette sales taxes provide 11.6 percent of total taxes. Involved in a deep economic crisis, the government relies on this revenue and has not introduced a single measure to control or counteract the high-pressure marketing by manufacturers. However, cigarette-related diseases far outnumber infections as the leading cause of death, in a developing country with a very large young population. While some medical associations have recently urged the government to take stronger measures, physicians are reluctant to speak out. With specific legislation badly needed to regulate sales and advertisements, the first task must be to convince policy makers that aiding and abetting the cigarette companies is, in the long run, bad for the economy. Medical institutions are in a key position to put pressure on the government and the tobacco industry. Since physicians are expected to provide necessary health education, neglecting to act against smoking is seen as a type of malpractice.

84–0961. Madeley, J. **The Environmental Impact of Tobacco Production in Developing Countries.** *New York State Journal of Medicine* 83(13): 1310-1311, December 1983.

Besides health-related damage, tobacco production destroys trees and land; deforestation is currently the most serious environmental problem now facing the Third World. About half the tobacco grown worldwide is flue-cured in barns, requiring, conservatively, an estimated hectare of trees for each ton of tobacco. Roughly one of every eight trees cut is for curing tobacco. However, economic incentives make tobacco growing irresistible to farmers, resulting in deforestation and loss of protection to the land, severe soil erosion, silt-filled dams, and lost food production. Besides the harm to the land, farmers are threatened by the insecticides necessary for a successful crop. In the first 4 months of cultivation, at least 16 applications are needed, yet farmers often do not understand the instructions, cannot afford protective clothing, often do not have soap or access to medical care, and cannot prevent the pesticides from entering the streams and rivers. Encouragement is needed from the World Health Organization and the Food and Agricultural Organization for farmers to substitute other crops, although the ultimate issue is whether or not the governments of these countries are willing to help the farmers switch.

84–0962. Maxwell, J. C., Jr. **Cigarette Sales in Downspin.** *Tobacco Reporter* 111(1): 50-51, January 1984.

Total industry sales for 1983 are estimated at 590 to 597 billion units, a drop of 5 to 6 percent from the 1982 figure of 624 billion units. Annual consumption appears to be down at least 4 percent, and the snapback in consumption is not as buoyant as the industry had hoped. The adverse effects on sales are attributed to the doubling of the Federal excise tax, numerous state tax increases, and rising health concerns. Also, low-tar sales appear to have topped out: the 1983 market share is estimated at 53.2 percent, compared with a 1982 level of 58.8 percent. Cigarette companies are gradually moving tar content up by several milligrams, as happened in Germany. The industry is compensating for the reduced sales with cost control measures and layoffs. The market shares of the top 10 cigarette brands are discussed.

84–0963. Nath, U. R. **India's Shame. A War on Smallpox But a Welcome for Cigarettes.** *New York State Journal of Medicine* 83(13): 1320-1321, December 1983.

Tobacco is a valuable source of revenue in India and little has been done to restrict manufacturers or educate the public on the health effects of smoking. Although health warnings are required on any "visible" representation, such warnings are in English and Hindi, rather than in the 16 regional languages, a patently false observance of the letter of the law. Successful promotional gimmicks and giveaways encourage sales in villages so remote that even government food distribution sometimes flounders. There is little public debate or consumer interest in health education against smoking, and only feeble attempts have been made by the government. With India's fast-growing population, educators are trying to design curricula to teach young people about birth control, nutrition, and other aspects of health, while the tobacco industry looks forward to a reservoir of advertising-indoctrinated youth. With a severely strained health care budget, health professionals and workers must be made aware that smoking is a cause of disease and that the costs and consequences of the smoking epidemic can be avoided by doing something about it now.

84–0964. New York State Journal of Medicine. **The Business of Smoking.** *New York State Journal of Medicine* 83(13): 1324-1332, December 1983.

Profiles of the six major American cigarette companies (American Tobacco, Brown and Williamson, Liggett and Myers, Lorillard, Philip Morris USA, and R. J. Reynolds, each part of a parent corporation) are presented. Tabular data for the tobacco subsidiaries include sales and market share for 1982, number of employees, 1982 advertising outlay, organizational profile, U.S. cigarette brands, advertising theme and agencies, and sponsorships/promotions. Corporate histories are given for each tobacco company. Sales in 1982 for the six parent companies were 42.8 billion dollars, and advertising expenditures amounted to 1.5 billion.

84–0965. Scott-Malden, N. **UK Tobacco Industry Update. The Challenge Facing the UK Tobacco Manufacturers.** *Tabak Journal International* 5: 398-400, 403, October 1983.

Rising cigarette taxes, growing unemployment, severe recession, and increasing health awareness have caused a sharp downturn in cigarette consumption in Great Britain, but price is considered the most important factor in diminishing sales. In the past 10 years, the cost of a typical king-size brand has increased by nearly 270 percent compared with the Retail Price Index, which rose 260 percent. Between 1973 and 1983, the proportion of smokers declined from 52 to 38 percent in men and from 41 to 33 percent among women, while cigarette sales fell 27 percent. In 1950, tobacco products accounted for 8.2 percent of total consumer expenditures, compared with only 3.6 percent in 1982. Although a continual decline in sales volume appears to be a permanent feature of the market, the 1983 cigarette tax increase is limited to 3 p/pack and sales are expected to fall only 2 percent this year. The industry is responding with cost controls (lost jobs, factory closings, and equipment investments for increased production) and increased exports to offset the domestic fiscal environment. However, the threat of generics and shifts in consumer tastes have prompted market upheavals through new brand launchings and price wars. The effects of entry into the European Common Market and the necessary harmonization of the tobacco duty system are also discussed.

84–0966. Stone, M. **Spotlight on the United Kingdom: A Bit of Buoyancy.** *Tobacco Reporter* 110(12): 44, 46, 48, December 1983.

While antismoking propaganda has played a small part in declining sales, and the government receives almost 80 percent of the price of a pack of cigarettes in taxes, the tobacco industry is expected to survive, but in a new form: fewer companies and customers, and fewer brands. The steady decline in cigar sales appears to be bottoming out and, at the end of a difficult year, there is a somewhat improved picture for cigarettes, too. New longer-than-king-size cigarettes were successful in 1983, and generic brands have had little impact on the general public, indicating that the customer has more confidence in established quality brands. Promotional activity is increasing, particularly in the cigar market, and sponsorship of sports activities continues. A spokesman believes the future health of the industry depends on the fiscal treatment by the government, which was based on the rate of inflation in 1983. Similar treatment is hoped for in 1984.

84–0967. Tabak Journal International. **Statistics.** *Tabak Journal International* 5: 423-424, October 1983.

Worldwide cigar production, import, and export data are presented. Unit volume information for the years 1979 through 1982 (and in some cases March 1983) is given in tabular form. Import data are provided for 33 countries, export data for 19 countries, and production data for 17 countries. The Netherlands and Belgium-Luxembourg showed the largest volumes of production, exports, and imports. Statistics for the United States show that exports increased from 177 to 354 million pieces between 1979 and 1980, but dropped to 181 million pieces in 1982. Import data indicate a slight increase, from 112 million pieces in 1979 to 119 in 1982. Production, however, has declined steadily, from 3,601 million pieces in 1979 to 3,195 in 1982.

84–0968. Tabak Journal International. **Cigars Poised to Do Well When British Recession Ends.** *Tabak Journal International* 5: 404-405, October 1983.

British cigar makers and importers expect that when the present economic depression lifts, sales will improve faster than those of other tobacco products. Between 1980 and 1982, cigarette sales fell 16 percent while cigars declined only 9 percent, indicating cigar smokers may be more loyal. Cigar traders are using the lull in business activity (cigar sales may reach only a little more than 1,400 million) to improve the social acceptability of cigar smoking. While making special efforts to promote sales in hotels, restaurants, and clubs, the manufacturers are also making use of their right to promote cigars through television advertising, which is forbidden for cigarettes. Industry critics have denounced cigar advertising because a few cigars carry the same brand name as cigarettes. However, the most insoluble problem comes from an unexpected source: Britain's cartoonists insist on putting fat cigars in the mouths of people depicted as rich. (Auth. Abs. Mod.)

84–0969. Tabak Journal International. **Cigar Market Continues to Drop.** *Tabak Journal International* 5: 380, October 1983.

Cigar sales in the United States continued to drop in 1982, and well into the first half of 1983. Consumption was off 5.2 percent in 1982 and 5.7 percent thus far in 1983. Premium cigars, however, were once again the bright spot of the industry, with steadily rising sales. The overall consumption decline, as in the past, is blamed on changing consumer habits, trends toward nonsmoking in public areas, continuing bad publicity, and a general decrease in the prime target age group for cigar smoking. Industry analysts hope that with the increasing age of the baby boom generation, cigar sales will once again pick up. (Auth. Abs.)

84–0970. Tabak Journal International. **Tobacco as an Important Source of Foreign Exchange for the Developing Countries.** *Tabak Journal International* 5: 364, 366-367, October 1983.

Both developing and industrial countries depend on tobacco exports for a substantial part of their foreign exchange earnings (up to half, as in Malawi) and as an important source of fiscal revenue (12 percent in India, 13 percent in the United States). Most governments encourage tobacco production with price support systems and control of plantings to stabilize farmers' incomes. Tobacco generates rural employment and is a significant source of agricultural wages. For the Zimbabwe farmer, for instance, tobacco earns twice as much as sugar, five times as much as cotton, and 10 times as much as maize. Of all crops grown, tobacco ranks eighth in value in the United States, fifth in Canada, fourth in Japan, seventh in Yugoslavia, and ninth in Turkey and Thailand. Of the world's six principal tobacco-producing countries, three are in the developed and three in the developing world. Although tobacco is one of the world's most widely grown crops, it takes up only 0.3 percent of the world's arable land.

84–0971. Teoh, S. K. **Smoking in Malaysia. Promotion and Control.** *New York State Journal of Medicine* 83(13): 1317-1319, December 1983.

Although tobacco was only introduced as a cash crop in Malaysia in 1959, it has become a major source of income for farmers, manufacturers, and the government (through taxes). Advertising has been the main promotional effort, but in 1982, all cigarette ads on TV and radio, and in government publications, were prohibited. Emphasis has shifted to newspapers and sponsorship of sports events; however, action is being taken to ban all cigarette advertising, including such sponsorship. Consumption (primarily by males) has risen, but an excise tax in 1982 led to a drop in sales. Other actions being targeted include (1) publication of tar, nicotine, and carbon monoxide levels; (2) stricter implementation of nonsmoking areas in public places; and (3) greater efforts in health education, especially in the non-English language media and with youth.

84–0972. Tobacco Reporter. **Spotlight on the United States: The Long, Hot Summer of '83.** *Tobacco Reporter* 110(11): 58-63, November 1983.

U.S. tobacco output may decline as much as 31 percent from 1982 levels, due to lower quotas, high temperatures, and prolonged dry weather. Also, cigarette consumption will drop about 3 percent for the second consecutive year. Approximately 307 billion cigarettes were smoked during the first 6 months of 1983 (more than half of these were low-tar, low-nicotine), roughly 14 million fewer than during the same period last year. Cigarette output is expected to decline by about 9.2 billion pieces (1.325 percent). Increasing Federal and state tax burdens, combined with other antismoking measures, are cited as contributing factors. The decline in consumption of large cigars, cigarillos, and little cigars is expected to continue. Pipe and roll-your-own tobacco will also maintain a slight downward trend. Data on output of U.S. tobacco products, and for U.S. leaf exports (by type and destination) are charted for 1980 through the 1983 estimates. Crop trends for different varieties and amendments to the tobacco program currently under debate in the House of Representatives are outlined.

84–0973. Tobacco Reporter. **World Leaf Trade: Exports and Imports Show Declines.** *Tobacco Reporter* 110(12): 38, 40, 42, December 1983.

In a year of large leaf surpluses, falling consumption, and generally poor economic conditions around the world, it is not surprising that overall trade in unmanufactured tobacco declined in 1982. All indications point to a further 2 percent drop in 1983. Import and export data are provided for the following major trading countries: Argentina, Brazil, Canada, Greece, India, Italy, Japan, Korea, Philippines, Spain, Switzerland, Thailand, Turkey, United Kingdom, United States, West Germany, Yugoslavia, and Zimbabwe. Imports and exports were down in most countries although exports were up by 56 percent in Argentina, 28 percent in Italy, 12 percent in Brazil, and 4 percent in Thailand. Reductions in imported unmanufactured tobacco were as high as 31 percent in Zimbabwe, 20 percent in Turkey, 18 percent in Greece, and 17 percent in Korea. In the United States, exports fell 2 percent and there was a 7 percent decrease in imports.

84–0974. Tobacco Reporter. **Spotlight on West Germany: Rough Road to Recovery.** *Tobacco Reporter* 110(12): 50, 52, December 1983.

The 39 percent tax increase of June 1982 resulted in a 17 percent drop in sales of traditional name-brand cigarettes for the year. Overall consumption, however, fell only 6 percent (to 138 billion pieces) as sales of roll-your-own and generic cigarettes soared to 22.3 billion pieces. Consumption of imports was only 8.3 billion. For the first 9 months of 1983, cigarette sales reached 83,957 million pieces, a decline of only 0.8 percent from the same period last year, while sales of fine-cut tobacco rose 3.6 percent to 13,319 metric tons. During autumn 1982, fine-cut consumption increased 80 percent, but since spring 1983 there has been a slight return to manufactured cigarettes. The abrupt shift in market patterns, and lowered consumption, has prompted the introduction of bargain brands, price-cutting maneuvers, and fierce competition among manufacturers. Market share trends among the leading brands are discussed.

84–0975. Tobacco Reporter. **Light Leaf Report: Flue-Cured and Burley Register Declines.** *Tobacco Reporter* 110(12): 54, 56, December 1983.

Flue-cured and Burley tobacco production reports are provided for Zimbabwe, Brazil, Thailand, Argentina, Korea, Canada, Italy, India, Malawi, Pakistan, and the United States. Overall world production declined due to unfavorable weather and high stock levels, which discouraged plantings. However, early reports from the Southern Hemisphere indicate that farmers plan to increase 1984 production to regain some of the loss suffered with the 1983 crops. Output of flue-cured for the year is estimated at around 3 million metric tons, and Burley has dropped to an estimated 718,000 metric tons.

84–0976. Warner, K. E. **The Economics of Smoking: Dollars and Sense.** *New York State Journal of Medicine* 83(13): 1273-1274, December 1983.

The annual smoking-related death toll is approximately 350,000 lives, and the annual economic per capita cost of smoking is 200 dollars. Smoking-related disease consumes 15 billion dollars/year in medical care resources (>5 percent of the nation's health care costs) and causes a productivity loss of 34 billion dollars/year. In contrast, consumers spent 25 billion dollars on cigarettes in 1982, 40 percent of the total spent on new automobiles. Tobacco accounts for roughly 2.5 percent of the gross national product, annually contributing 60 billion dollars and 2 million jobs to the economy. Such economic muscle has been reflected in anemic mass media coverage of smoking and health issues, and for decades major Federal antismoking legislation was unthinkable. However, antismoking forces have banded together in a lobby, and a "no net cost" bill has been passed into law. More legislation concerning strengthened health warnings, passive smoking, and other issues is expected.

LEGISLATION

84–0977. Burchard, L. L. **Clean Indoor-Air Legislation: The Illinois Experience.** *New York State Journal of Medicine* 83(13): 1303, December 1983.

In the first attempt to legislate clean indoor air measures in Illinois in 1974, the bill was passed by the general assembly but was vetoed by Governor Dan Walker, who claimed there was no provision for enforcement. Legislation that provided enforcement was passed in 1978, only to be vetoed by Governor Thompson within minutes after he met with cigarette company lobbyists. In 1980, a bill was stopped by the Department of Public Health, which claimed enforcement would be too costly. A 1983 bill was written that took all previous objections into account and had wide support from the medical community. Unexpectedly, the bill was opposed by many black and female legislators, who, it is noted, are currently primary targets of advertising and a fast-growing market for cigarettes. With one exception (a tobacco lobbyist), all witnesses against the bill were from out-of-State, and all were paid by the tobacco industry. The bill was sent to a subcommittee composed entirely of senators opposed to the legislation. Even so, grassroots support is growing that, hopefully, will be translated into legislation.

84–0978. Jauvtis, R. L. **The Rights of Nonsmokers in the Workplace: Recent Developments.** *Labor Law Journal* pp. 144-148, March 1983.

Courts and arbitrators are adjudicating a growing number of cases dealing with the rights of nonsmokers in the workplace. The cases usually arise as matters of first impression, stemming from increasing public awareness of the hazards of cigarette smoking (mainstream smoking) and of inhaling secondary tobacco smoke (sidestream smoke). Since the Occupational Safety and Health Administration has not promulgated a specific safety standard on permissible exposure levels (ostensibly because tobacco smoke is not a necessary byproduct of any manufacturing process), the cases are predicated upon the employer's common law duty to provide employees with a safe workplace. Details of the earliest litigation, a 1976 suit against the New Jersey Bell Telephone Company, are reviewed, along with several subsequent rulings and arbitration decisions. In general, the cases indicate an employer must seriously attempt to accommodate employees who complain about severe adverse medical reactions to tobacco smoke. In suits involving a unionized work force, a blanket no smoking rule (which would include areas with no evident fire hazards) will probably not be upheld unless there are very specific and compelling reasons.

84–0979. Kahn, P. L. **The Minnesota Clean Indoor Air Act. A Model for New York and Other States.** *New York State Journal of Medicine* 83(13): 1300-1301, December 1983.

While the 1975 Minnesota Clean Indoor Air Act was not the first law to attempt to control smoking in public places, it was the first to prohibit smoking everywhere unless it was specifically permitted. The law, which has been lauded by health professionals but criticized by the tobacco lobby as restrictive, confusing, and the first step in the total destruction of personal freedoms, was favored by 92 percent of nonsmokers and 87 percent of heavy (two packs/day) smokers in the 1980 Minnesota poll. Although the original objective was protection of clean indoor air rather than individual smoking deterrence, as smoking has become socially and legally unacceptable, and as it has become more difficult to smoke, cigarette consumption has declined. Results of a 1978 Roper Organization poll indicate that the nonsmokers' rights movement is "the most dangerous development to the viability of the tobacco industry that has yet occurred." Data show that the tobacco industry loses 500 million dollars/year if each American smokes one less cigarette/day. Factors contributing to passage and enforcement of the law are given, and local and national organizations working actively on behalf of nonsmokers' rights are listed.

84–0980. Tate, C. F., Jr. **A Physician-Led Referendum for Cleaner Air in Florida.** *New York State Journal of Medicine* 83(13): 1302, December 1983.

After repeated futile attempts to get the Dade County Commission to pass a Clean Indoor Air Act, the issue was put directly before the public as a referendum in 1979. The referendum was defeated by only 820 votes. It was later revealed that more than 1 million dollars was spent in mass media advertising to defeat the measure, the majority coming from tobacco companies; less than a thousand dollars was contributed by Dade County residents. Supporters of the measure had only 5,000 dollars to spend. The Dade County Medical Association went on record for the ordinance, but was the only major health organization to offer support. The local chapter of GASP (Group Against Smoking Pollution) provided support, including help in obtaining the signatures needed to get the measure on the ballot. The effort was repeated in 1980, with hopes that the voluntary health agencies and the medical community would join the battle. They did not, however, and the referendum was again defeated. The opposing effort put forth by the tobacco industry is described, and the necessity for support from the medical community is emphasized.

84–0981. Teret, S. P.; DeFrancesco, S. **The Cigarette Safety Bill: A Case Study in Injury Control Advocacy.** *Journal of Public Health Policy* 4(4): 440-446, December 1983.

A prototype bill, introduced in the Maryland State Legislature, aimed at controlling fires caused by cigarettes, is analyzed. Ten such bills, which vary somewhat in content (particularly in terms of who is authorized to promulgate the standard for cigarettes and little cigars that would lower their propensity to ignite housefires), have been introduced in other State legislatures, and in the United States Congress. Provisions of the Maryland Act would (1) require fire safety standards for cigarettes and small cigars sold in the State; (2) require standards to specify that cigarettes and little cigars self-extinguish or not ignite upholstered furniture or mattresses; (3) provide that those not meeting the standards may not be sold after a certain date; (4) define certain terms; (5) provide for the seizure of certain cigarettes and little cigars and for the enforcement of the Act; and (6) include provisions generally relating to fire safety standards for cigarettes and little cigars. Discussion of the bill is aimed at making the reader aware of the process involved in drafting public health legislation that can directly result in the prevention of death and injury.

84–0982. U.S. Department of Health and Human Services. **State Legislation on Smoking and Health, 1982.** U.S. Department of Health and Human Services, Public Health Service, Office of the Assistant Secretary for Health, Office on Smoking and Health, July 1983, 92 pp.

The 235 bills and 35 laws reported for 1982 are presented in five tables: (1) summary, by State, of legislation introduced and passed in seven categories (Limitations, Commerce, Schools, Advertising, Sales to or Use by Minors, Insurance, Other); (2) bills passed into law in 1982 by subject and State; (3) a State-by-State breakdown of bills introduced and passed, and resolutions introduced and adopted; (4) laws passed pertaining to smoking and health that have been reported in *State Legislation on Smoking and Health* for 1975 through 1982; and (5) a quick reference to the level of nationwide smoking and health legislative activity, listing the number of smoking and health bills introduced and passed from 1975 to 1982. For 1982, 4 of 54 bills introduced under Limitations were passed into law by California, Hawaii, and New Jersey; 21 States passed 29 bills in the Commerce category; one bill in the Schools category was enacted in California; Idaho passed one bill in the Sales to or Use by Minors category; and no bills were enacted in the Advertising or Insurance categories.

See also, **84–0988**

GENERAL

84–0983. Burney, L. E. **Policy Over Politics. The First Statement on Smoking and Health by the Surgeon General of the United States Public Health Service.** *New York State Journal of Medicine* 83(13): 1252-1253, December 1983.

While some progress has been made in the fight against smoking in the 26 years since the first statement by the Surgeon General (implicating smoking as a causative factor in lung cancer), the battle is far from won. Special economic interests and the politicians they influence still refuse to drop their membership in the Flat Earth Society and acknowledge that cigarette smoking is killing hundreds of thousands of Americans each year. The early evidence on which the first report was based, and the accumulated evidence now available, are reviewed. The changing attitudes among physicians, who are taking more positive and direct action in informing the public and influencing the smoking habits of their patients, are commended. This role must be continued to reduce and prevent the mortality and disability from lung cancer, emphysema, heart disease, and other diseases in which cigarette smoking has a causative role.

84–0984. Cleary, A.; Shelley, E. **Smoking and Health - The Facts in Ireland.** *Irish Medical Journal* 76(12, Supplement): 1-29, December 1983.

Four aspects of the smoking problem in Ireland are discussed: smoking patterns; smoking-related health problems; current smoking control measures; and cessation techniques. The review of smoking patterns includes a history of tobacco use in Ireland, and recent patterns and current trends in cigarette consumption according to age, sex, and social class. Current measures of tobacco control involve use of health warnings, limitations on yields of smoke components, taxation, restrictions on sales and promotion, and bans on smoking in certain public places. Smoking-related health problems include cardiovascular diseases, cancer, bronchitis, emphysema; the effects of passive smoking and maternal cigarette use during pregnancy; and overall morbidity and mortality related to smoking in Ireland. Cessation techniques currently in use involve clinics, medication, hypnosis, educational methods, and mass media programs. Two appendixes provide (1) a bibliography of relevant Irish research and a list of journals that publish smoking and health research; and (2) names and addresses of organizations conducting antismoking activities.

84–0985. Cromer, M. A. **"Precious Baby".** *New York State Journal of Medicine* 83(13): 1292-1293, December 1983.

It is naive to suggest that smoking by a pregnant woman is a decision made by an informed adult. Survey data have repeatedly demonstrated that, when compared with nonsmokers or ex-smokers, persons who smoke are ignorant of the severity and probability of cigarette-caused damage. The media, especially magazines aimed at women, have done little to educate. Journals that claim to promote the status of women censor articles about smoking and health and continue to carry many pages of cigarette advertising. One magazine, which recently devoted an entire issue to the topic of women's health, has never published an article on smoking. Women's medical, civic, and religious organizations are encouraged to do more to stop the cigarette advertising assault on females. In the long run, however, social unacceptability and the increasing involvement of women in nonsmokers' rights groups provide hope that publishers of exploitive magazines will find fewer and fewer women buying such publications.

84–0986. Femi-Pearse, D. **Aspects of Smoking in Developing Countries in Africa.** *New York State Journal of Medicine* 83(13): 1312-1313, December 1983.

Governments in Third World countries have been slow to discourage the smoking habit because of the high revenues associated with tobacco production. Also, the benefits of tobacco cultivation to the farmer cannot be dismissed: the cash returns are better than for food crops, and the farmers are prosperous and enjoy higher status. The tobacco industry provides training in modern farming skills, offers scholarships to the children, extends credit for farm development, and explains new marketing strategies and international business practices. Tobacco cultivation has serious environmental and economic effects, however, and to win the fight against tobacco, Third World governments must sustain growth of food crops via land preparation, distribute seed and fertilizer, and provide stable prices. It is noted that the currently high smoking rates among both adults and children will soon assume an enormous economically negative health importance.

84–0987. Gitlitz, G. **Cigarette Advertising and *The New York Times*: An Ethical Issue That's Unfit to Print?** *New York State Journal of Medicine* 83(13): 1284-1291, December 1983.

Correspondence over the past 7 years, between a vascular surgeon and *The New York Times*, concerning the paper's cigarette advertising policy is presented. The surgeon's letters criticize the presence of cigarette ads in *The Times*, cite the health consequences of smoking as adequate reason to refuse advertising, and challenge them to discuss on the editorial page the reasons for continuing to accept advertising for cigarettes (including a financial statement). The few replies by the paper, generally from the advertising acceptability department, point out that the harm due to smoking, and research developments concerning cigarettes, have been repeatedly printed. However, it is argued that cigarettes are a legal product and advertising that meets *The Times'* standards is accepted just as the decision to smoke is ultimately made by informed adults.

84–0988. Grannis, A. B. **The New York Cigarette Fire Safety Act.** *New York State Journal of Medicine* 83(13): 1299, December 1983.

Cigarettes are the leading cause of residential fire mortality in the United States, but the tobacco industry claims that the public would not wish to smoke any existing form of firesafe cigarette. The industry, while insisting that cigarettes pose no health risks regardless of tar and nicotine content, expresses concern that firesafe cigarettes would raise tar and nicotine levels. However, the National Bureau of Standards indicates no correlation between tar and nicotine yields and a cigarette's propensity to ignite furniture. Tobacco companies' opposition to regulation is apparent in efforts to stop (1) the Cigarette Fire Safety Act, which would require the New York State Office of Fire Prevention and Control to set fire safety standards for cigarettes sold within the State; and (2) Assembly Bill 939, which would require cigarette companies to provide the Commissioner of Health with a list of chemical substances added to their products. While the tobacco industry has managed to exclude cigarettes from classification as a food, drug, or hazardous substance, chemical components in both the gas and particulate phases of cigarette smoke (i.e., toluene and vinyl chloride) have widely ranging physiological effects. The six major cigarette manufacturers currently disclose ingredients that are added by three or more companies in self-determined "substantial" amounts.

84–0989. Kusinitz, M. **"Non-Smoking, Please." A Money-Saving Proposition.** *New York State Journal of Medicine* 83(13): 1361-1363, December 1983.

Businesses that cater to nonsmokers are proliferating, and include airlines, travel clubs, restaurants, car rental agencies, hotels and motels, and insurance agencies. Many industries are restricting smoking to places like restrooms, or separating smoking from nonsmoking workers. Employment agencies increasingly receive requests for people who do not smoke. Besides the gratitude of patrons and employees, businesses that ban or restrict smoking receive benefits in savings on insurance premiums, cleaning and repair bills, energy costs from lowered air circulation, and increased productivity. The tobacco industry, aware of the threat from the attitude that smoking is socially unacceptable, has countered the antismoking stance with claims of undermined personal freedom while glorifying the hard-driving worker who smokes. However, nonsmokers comprise two-thirds of the U.S. work force, and the majority object to smoking on the job.

84–0990. Lancet. **Third World Smoking—The New Slave Trade.** *Lancet* 1(8367): 23-24, January 7, 1984.

The rapid spread of the smoking epidemic to Third World countries is attributed to commercial enterprise, government inactivity, and encouragement of tobacco companies based in the United Kingdom and United States. British governmental support of both the tobacco industry and tobacco exports to developing countries is outlined. Smoking-related diseases cataloged in World Health Organization (WHO) reports for 1978 and 1983 include high lung cancer mortality in India, China, Hong Kong, Cuba, and the Bantu of Nepal; coronary heart disease in India, Pakistan, and the Philippines; doubled perinatal mortality rates among Bangladeshi women who smoke; and oral cancer among Asian tobacco chewers. Developing countries are urged to give high priority to smoking control activities. WHO recommendations include smoking control agencies in individual countries, cigarette sale prohibitions and health education for children, tobacco advertising bans, cost—benefits analysis, alternative crops, and more stringent governmental controls in developed countries. Smoking control activities are suggested for British doctors.

84–0991. Lessof, M. H. **Culpable Negligence: A Case for the Courts?** *New York State Journal of Medicine* 83(13): 1282-1283, December 1983.

Cigarette consumption in the United Kingdom fell 16 percent between 1960 and 1980, followed by a 9 percent decline in 1981 and a 7.5 percent fall in 1982. Although the steps taken by the government against the tobacco industry have been feeble, smoking is becoming socially less acceptable, and the growing unpopularity of the tobacco industry is being translated into political pressure. Government action is still needed on two fronts: a ban on all tobacco promotion and an increase in taxes. The tobacco lobby has fought back with gift-coupon schemes, health research donations (that must avoid investigation of tobacco), television coverage of tobacco-sponsored sporting events, art and travel promotions, and claims that advertising bans would cause unemployment and close down newspapers. Norway's advertising prohibitions and the resulting decreases in consumption are reviewed, and it is noted that the evidence is strong enough to convince any impartial judge or jury of the role of cigarettes in causing death and disability.

84–0992. McGuire, A. **Cigarettes and Fire Deaths.** *New York State Journal of Medicine* 83(13): 1296-1298, December 1983.

Fires attributed to cigarettes (the major cause of fire death) kill over 2,300 persons and burn an additional 5,000 annually in the United States. In 1981, 65,000 fires started by cigarettes resulted in 300 million dollars in property damage. Of 530 fire fatalities in Maryland from 1971 to 1977, 45 percent were cigarette-related (compared with 8 percent attributed to heating equipment). Prevention methods suggested include (1) warning smokers not to smoke in bed and to properly dispose of cigarette butts; (2) creating a more fire-resistant environment, an approach favored by the tobacco industry; and (3) modifying cigarettes, which the tobacco industry claims is not possible. Nevertheless, studies show that firesafe cigarettes are feasible, and over 95 patents have been issued worldwide for methods that render cigarettes firesafe or self-extinguishing. Firesafe cigarette legislation introduced on both Federal and state levels is discussed; conclusions of researchers at the National Bureau of Standards regarding safer cigarette construction are listed; and funds and equipment contributed by the Tobacco Institute and individual cigarette companies (i.e., Philip Morris), most frequently in states considering fire safety legislation, are described. Areas of activity planned for 1984 include expansion of the coalition that will lobby State capitals and Washington, D.C.; an international firesafe cigarette campaign; and personal injury and product liability suits filed on behalf of victims.

84–0993. Pertschuk, M. **The Politics of Tobacco: Curse and Cure.** *New York State Journal of Medicine* 83(13): 1275-1277, December 1983.

Resistance to national antismoking measures is buttressed by the nation's economic dependence on tobacco revenues, by workers who depend on the industry for a living, and by smokers themselves. The tobacco industry, although rich and powerful, is vulnerable to astute political action, and even sees itself as the victim of the public health establishment and regulators' hunger for power. However, the objective of the antismoking force is the reduction of smoking-related disease, not retribution against those with an economic stake in tobacco. It is argued that removal of the price support program would result in poor tobacco farmers, but not a weakened tobacco industry or less smoking-related morbidity. The ultimate and most effective weapon against smoking will be the combined force of solid scientific expertise, community organizations of concerned professionals and public health charities, political leaders, a network of lobbyists, and support from a mass media unfettered by advertising dollars.

84–0994. Richards, J. W., Jr. **A Positive Health Strategy for the Office Waiting Room.** *New York State Journal of Medicine* 83(13): 1358-1360, December 1983.

A survey by Doctors Ought to Care (DOC) found that only three of the ten most subscribed-to magazines by physicians for their waiting rooms refused cigarette advertising: *National Geographic, Good Housekeeping*, and *Readers Digest*. One physician reported 390 pages of cigarette ads in his waiting rooms. Cigarettes are the most advertised product in the magazines most often found in waiting rooms, comprising 15 to 80 percent of advertising/issue in magazines such as *TIME* or *Newsweek*. Most of the physicians surveyed were aware that smoking is the most frequent cause of premature death in the United States, and most expressed a need for more patient education materials. Although 70 percent felt the waiting room is a good setting for health education, only 18 respondents spent >60 dollars/year on materials, while two-thirds spent >50 dollars/year on magazine subscriptions. The American Medical Association recently congratulated a group of publishers for refusing to accept cigarette advertising. A list of magazines that do not carry cigarette ads is included.

AGRICULTURE

84–0995. Mylonas, V. A.; Ntzani, S. H. **Effect of N and K Fertilization on Agronomic Characteristics and Chemical Composition of Myrodata of Agrinio Oriental Tobacco.** *Tobacco International* 186(2): 37-39, January 20, 1984.

Tobacco yield increased significantly as nitrogen (N) applications were increased from 20 to 80 kg/ha, and value increased as potassium (K) was increased from 30 to 90 kg/ha. Field experiments were conducted on sandy loam soils for 3 years (1973 to 1975) at Makryneia of Agrinio, Greece, to determine the response of oriental tobacco (*Nicotiana tabacum* L. cv. MA 13/b) to four N rates (20, 40, 60, and 80 kg/ha) and to four K rates (30, 60, 90, and 120 kg/ha). The tobacco burn rate and total alkaloids, total N, calcium (Ca), and magnesium (Mg) concentrations in cured leaves increased as nitrogen rates increased. Concentrations of K in leaves increased as applied K increased. Rates of K had no significant effects on burn rate and concentrations of total alkaloids, total N, Ca, and Mg in cured leaves. Results suggest that 20 to 40 kg N/ha, or in some cases 20 to 60 kg N/ha, and 60 to 90 kg K/ha, depending on soil fertility, are adequate for high yield of good quality oriental tobacco in this region. (Auth. Abs.)

84–0996. Zinninger, T. C.; Deli, J. **Composition and Method for Controlling Growth of Tobacco Suckers.** United States Patent No. 4,404,016, September 13, 1983, 3 pp.

A method for controlling the growth of tobacco suckers is described that utilizes a liquid composition containing an alkylene glycol solution of alkyl N-(halophenyl)carbamate (e.g., isopropyl N-(3-chlorophenyl)carbamate), which is applied to topped tobacco plants that have previously been treated with a contact sucker control agent. The quantity of alkyl N-(halophenyl)carbamate in the composition may vary over a wide range (from about 1.0 percent by weight up to the maximum solubility of the carbamate in the alkylene glycol). Preferably, the composition contains from about 12 to 14 percent carbamate by weight based on weight of alkylene glycol, and is applied about 7 to 10 days after the systemic sucker control agent, effectively inhibiting further sucker growth.

CHEMISTRY

84–0997. Angenot, L. **Composition Chimique de la Fumee de Tabac. [Chemical Composition of Tobacco Smoke.]** *Revue Medicale de Liege* 38(17): 632-640, September 1, 1983, French.

Various types of tobacco are grown and processed according to climatic conditions and practical agricultural considerations in many countries, and usually mixed for smoking products; a percentage (in Belgium >20 percent) of other products is added to give a distinctive flavor. Experimental results are discussed and tabular data provided on the chemical components of cigar and cigarette smoke. Only major components are included, since over 1,200 substances have thus far been identified in tobacco and its combustion products. Tobacco smoke is a concentrated aerosol of liquid particles suspended in an atmosphere consisting mainly of nitrogen, oxygen, and carbonic anhydride. The exact composition of the particulate phase depends on tobacco characteristics and smoking techniques. At times other very dangerous components (tar, benzopyrene, nicotine, and carbon monoxide) are found. Studies on tobacco smoke constituents have resulted in the reduction of certain toxic components, including tar (reduced from 28 mg to 1 mg), nicotine (from 1.7 to 0.1 mg), carbon monoxide (from 18 to 2), nitrogen oxide (from 0.3 to 0.03), hydrocyanic acid (from 0.4 to 0.01), and acrolein (from 0.4 to 0.01 mg). Higher values reflect commercial nonfiltered cigarettes; lower figures the "super low-tar" varieties.

84–0998. Brunnemann, K. D.; Masaryk, J.; Hoffmann, D. **Role of Tobacco Stems in the Formation of *N*-Nitrosamines in Tobacco and Cigarette Mainstream and Sidestream Smoke.** *Journal of Agricultural and Food Chemistry* 31(6): 1221-1224, November-December 1983.

Nine experimental cigarettes without filter tips were prepared with 70 percent of a defined blend of laminae and 30 percent of various types of stems, resulting in nitrate contents between 0.68 and 2.65 percent. *N'*-Nitrosonornicotine (NNN, 1.02 to 6.35 ppm) and the sum of tobacco-specific *N*-nitrosamines (TSNA, 1.95 to 8.57 ppm) were found to be strongly correlated with the nitrate content of tobacco ($r^2=0.978$ and 0.948). The yields of total particulate matter, nicotine, carbon monoxide, and carbon dioxide in mainstream smoke are not greatly influenced by the nitrate content; however, *N*-nitrosodimethylamine (NDMA) and NNN are correlated with the nitrate content of the tobacco ($r^2=0.957$ and 0.889). The sidestream smoke of six of the nine cigarettes delivered exceptionally high amounts of NDMA (>1000 ng/cigarette). The yields of NDMA and NNN in sidestream smoke were correlated with the nitrate content of the blend ($r^2=0.501$ and 0.767); however, other factors influence the nitrosamine yields in sidestream smoke. (Auth. Abs.)

84–0999. Federal Trade Commission. **"Tar," Nicotine and Carbon Monoxide of the Smoke of 207 Varieties of Domestic Cigarettes.** Federal Trade Commission, February 1984, 22 pp.

Tabular data are provided on the tar (dry particulate matter), total alkaloid (reported as nicotine), and carbon monoxide content of 207 varieties of cigarettes tested via the Cambridge filter method. Samples were obtained by purchasing, when possible, two packages of each variety of cigarettes distributed by domestic manufacturers between August 1982 and December 1982 in 50 geographic locations throughout the country. In tests of the 207 varieties, 15 could be smoked to 23 mm; the butt lengths of the other 192 varieties ranged from 23.8 mm to an average of 41.5 to 43.0 mm. The butt lengths of 171 of the 207 varieties tested exceeded 30 mm. Tar and carbon monoxide content is reported to the nearest 0.1 mg and the nicotine content to the nearest 0.01 mg, each with appropriate statistical values. Cambridge, Carlton, and Now (king-size and 100 mm), all king-size filtered cigarettes in hard packs, registered <0.5 mg tar, <0.05 mg nicotine, and <0.5 carbon monoxide. English Ovals (king-size, nonfiltered, hard pack) registered the highest levels of tar at 29 mg/cigarette and nicotine at 2.1 mg/cigarette, but at 15 mg carbon monoxide/cigarette, 34 brands registered higher levels (Bull Durham was highest at 23 mg).

84–1000. First, M. W. **Environmental Tobacco Smoke. 1. Exposure Levels. 1.1. Environmental Tobacco Smoke Measurements: Retrospect and Prospect.** *European Journal of Respiratory Diseases* 65(Supplement 133): 9-16, 1984.

Characterization of cigarette smoke components depends on the condensing temperature, since the particulate phase and the gas phase of cigarette smoke are defined as the parts that are, or are not, trapped by a Cambridge filter. But because hundreds, and perhaps thousands, of the compounds in cigarette smoke are markedly affected by vapor pressure and dilution in addition to temperature, measurement of environmental tobacco smoke (ETS) is difficult. Smoke exhaled by the cigarette smoker also makes a significant contribution to ETS. One study showed that the smoke retained in the lungs varied from 22 to 75 percent. However, exhaled smoke is changed considerably by the respiratory tract environment. At present, there is no satisfactory method for measuring ETS. When carbon monoxide, particulates, and nicotine are measured simultaneously, there is poor correspondence in results, owing to (1) gas appliances and street traffic that influence carbon monoxide levels, (2) particulate concentrations that often reflect the number of people moving around, and (3) the wide variation in nicotine levels with respect to other cigarette smoke components.

84–1001. Ishizu, Y. **Determination of the Refractive Index of Individual Cigarette Smoke Particles.** *Journal of Colloid and Interface Science* 95(2): 586-588, October 1983.

Using a best-fit method, the refractive index (RI) of sidestream smoke was 1.50 to 1.55. There was no apparent dependence of the RI on particle size. The RI was slightly higher than that previously reported for mainstream, but more study is needed to determine the significance of this observation. Simultaneous determination of RI and particle size is subject to much larger error than single determinations. A schematic diagram of the light-scattering cell, typical patterns, and an error contour map is given.

84–1002. Jenkins, R. A.; Guerin, M. R. **Environmental Tobacco Smoke. 1.3. Analytical Chemical Methods for the Detection of Environmental Tobacco Smoke Constituents.** *European Journal of Respiratory Diseases* 65(Supplement 133): 33-46, 1984.

Single component analytical methods used to determine levels of environmental tobacco smoke (ETS) constituents (carbon monoxide, particulate matter, nicotine, nitrogen oxides, aldehydes, benzo(*a*)pyrene, nitrosamines) are reviewed, and passive samplers (diffusion and permeation) and their uses are described. Multicomponent chromatographic profiling techniques, which overcome some disadvantages of single component analytical methods for ETS, are schematically depicted. It is indicated that even with sequential chromatography, passive sampling, and multicomponent profiling, which more accurately characterize ETS, results must always be assessed for the potential influence of confounding factors from other, nontobacco-smoke sources.

84–1003. Rickert, W. S.; Robinson, J. C.; Collishaw, N. **Yields of Tar, Nicotine, and Carbon Monoxide in the Sidestream Smoke From 15 Brands of Canadian Cigarettes.** *American Journal of Public Health* 74(3): 228-231, March 1984.

Sidestream smoke yields for 15 brands of cigarettes were determined under conditions where mainstream yields were approximately equal to those used to determine the values that appear on packages of Canadian cigarettes. Sidestream yields of tar, nicotine, and carbon monoxide were much higher than mainstream yields for all brands tested. The average sidestream-to-mainstream ratios for the 15 brands were 3.5, 6.6, and 6.8 for tar, nicotine, and carbon monoxide, respectively. The highest sidestream yields were obtained from brands with the lowest mainstream yields. (Auth. Abs.)

84–1004. Walters, C. L. **III.I. General Introduction.** In: Egan, H.; Preussman, R.; Eisenbrand, G.; Spiegelhalder, B.; O'Neill, I. K.; Bartsch, H. (Editors). *Environmental Carcinogens. Selected Methods of Analysis.* Volume 6—*N*-Nitroso Compounds. Lyon, International Agency for Research on Cancer, IARC Publication No. 45, 1983, pp. 285-294.

The principles of the separation, cleanup, and determination of nonvolatile *N*-nitroso compounds, including those occurring in tobacco smoke, are reviewed. The nonvolatile *N*-nitrosamines are those that cannot be readily volatilized to any great extent in a stream of steam. The principal *N*-nitrosamines of this type in mainstream and sidestream smoke include *N'*-nitrosonornicotine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-(butanone), and *N'*-nitrosoanatabine. The means for the determination of *N*-nitroso compounds discussed include photolytic procedures, chemiluminescence techniques, and determination by denitrosation. Also, specific clean-up procedures are reported for the tobacco-specific nonvolatile *N*-nitrosamines, *N*-nitrosamino acids, *N*-nitroso-3-hydroxypyrrolidine, and *N*-nitrosodiethanolamine.

84–1005. Weber, A.; Fischer, T. **Schadstoffkonzentrationen im Blasfeld von Rauchern. [Concentrations of Pollutants in the "Blowing Cloud" of Smokers.]** *International Archives of Occupational and Environmental Health* 53(1): 47-50, 1983, German.

Concentrations of particulate matter (PM) and nitrogen oxide (NO) were measured in exhaled clouds of cigarette smoke. At 1 m from the smoker, peak values of NO ranged from 41 to 750 ppb, and for PM 3,330 to 99,680 μg/m³. Concentrations decreased very rapidly; the half life was between 2 and 20 sec. At 3 m from the smoker, the exhaled cloud could not be detected. Both sidestream smoke and the relatively high concentration of PM in exhaled smoke may account for the symptoms of acute irritation in the passive smoker. (Auth. Abs.)

84–1006. Wiethaup, W. **Apparatus for Determining the Flow Behavior of a Smokable Article.** United States Patent No. 4,400,972, August 30, 1983, 9 pp.

An apparatus for determining the flow behavior of a smokable cigarette is described, which permits the measurement of ventilation flows in the areas of different lengths of the cigarette. The apparatus is comprised of an air-tight chamber at least partially surrounding the cigarette, which can be moved on a holding means that holds the cigarette. Flow characteristic measuring devices are coupled to the holding device for measuring the flow characteristics of a gas flowing through the smokable article. (Auth. Abs. Mod.)

PHARMACOLOGY AND TOXICOLOGY

84–1007. Ator, N. A.; Griffiths, R. R. **Nicotine Self-Administration in Baboons.** *Pharmacology Biochemistry and Behavior* 19(6): 993-1003, 1983.

Two experiments were conducted with baboons in which responding maintained by nicotine and cocaine was studied under two different schedules of drug delivery. In Experiment 1, nicotine (0.01-0.32 mg/kg i.v.) was available under a fixed-ratio 2 timeout 15 sec reinforcement schedule. When nicotine was substituted for cocaine or saline, dose-dependent differences in self-administration were evident across the first five sessions, resulting in an inverted U-shaped dose—response curve. With continued exposure to each nicotine dose, however, number of injections generally stabilized at levels not very different, if at all, from saline; and the terminal dose—response functions generally were low and flat. In Experiment 2, nicotine (0.01-0.56 mg/kg i.v.) was available under a fixed-interval 5 min timeout 60 sec reinforcement schedule. Response rates were considerably lower and response patterning was less likely to be scalloped than when responding was maintained by either cocaine or food, but number of injections was higher than those maintained by saline. When fixed-interval value was varied, number of nicotine reinforcements remained low and virtually constant, but number of food reinforcements increased as the fixed interval decreased. Results suggest that the ability of nicotine to serve as a reinforcer appears to be strongly influenced by the conditions of drug availability, perhaps more so than for other drugs of abuse. (Auth. Abs.)

84–1008. Aviado, D. M. **Environmental Tobacco Smoke. 1.4. Carbon Monoxide as an Index of Environmental Tobacco Smoke Exposure.** *European Journal of Respiratory Diseases* 65(Supplement 133): 47-60, 1984.

Usefulness and limitations of carbon monoxide as an index of environmental tobacco smoke (ETS) exposure are reviewed. Results of controlled experimental situations that investigated mainstream carbon monoxide/cigarette, sidestream to mainstream carbon monoxide ratio, and equations for predicting carbon monoxide levels are discussed, as are results of controlled tobacco smoke exposure experiments analyzing both carbon monoxide and carboxyhemoglobin levels. Usefulness of carbon monoxide as an index of indoor tobacco smoke exposure in realistic situations such as offices, restaurants and public places, and vehicles is also assessed. Reported indoor carbon monoxide levels can be regarded as an index of ETS exposure provided other sources of carbon monoxide are recognized, measured, and deducted. While data suggest little reason to suspect adverse effects on human health from reported indoor carbon monoxide levels normally associated with ETS exposure, the general population should be protected from exposure resulting in carboxyhemoglobin levels of >5 percent. Additional studies should determine whether levels from 1.8 to 3.0 percent are harmful to certain population segments.

84–1009. Badawy, A.A.-B.; Evans, M. **Opposite Effects of Chronic Administration and Subsequent Withdrawal of Drugs**

of Dependence on the Metabolism and Disposition of Endogenous and Exogenous Tryptophan in the Rat. *Alcohol and Alcoholism* 18(4): 369-382, 1983.

Nicotine (1.0 mg/100 ml drinking water) was administered to male Wistar rats for 3 weeks, and the effects of withdrawal on tryptophan metabolism and related metabolic systems were analyzed on days 1, 2, 3, and 8. The initial nicotine administration decreased the total enzyme and apoenzyme activities of liver tryptophan pyrrolase by 47 and 70 percent respectively, but increased tryptophan, 5-hydroxytryptamine (5-HT), and 5-hydroxyindol-3-ylacetic acid (5-HIAA) by 14 to 23 percent. Subsequent nicotine withdrawal increased pyrrolase holoenzyme, total enzyme, and apoenzyme activities by 141, 153, and 157 percent, respectively. Conversely, concentrations of liver, free serum, total serum, and brain tryptophan, and brain 5-HT and 5-HIAA were decreased by 19, 18, 20, 17, 19, and 21 percent, respectively. Nicotine withdrawal also decreased the extent to which a tryptophan load elevated the above parameters. Changes correlated with administration and withdrawal of ethanol, morphine, and phenobarbitone are also documented.

84–1010. Buffoni, F.; Santoni, G.; Albanese, V.; Dolara, P. **Urinary Mercapturic Acid in Chemical Workers and in Control Subjects.** *Journal of Applied Toxicology* 3(2): 63-65, 1983.

Smoking habits were not a significant factor in urinary excretion of mercapturic acid among 41 male chemical plant (ink) workers. The subjects, aged 25 to 50 years, were compared to a control group of University of Florence clerical workers. Mercapturic acid excretion was significantly lower in the chemical workers (smokers 43.3±2.6 mg/day, nonsmokers 42.4±5.6) compared with controls (smokers 64.6±11.0 mg/day, nonsmokers 57.5±6.5). No correlation was found between daily mercapturic acid excretion and urinary mutagenic activity on strain TA 100. It is suggested that urinary thioether levels are not a good measure of exposure to toxic chemicals.

84–1011. Carney, R. M.; Goldberg, A. P. **Weight Gain After Cessation of Cigarette Smoking. A Possible Role for Adipose-Tissue Lipoprotein Lipase.** *New England Journal of Medicine* 310(10): 614-616, March 8, 1984.

Results suggest that lipoprotein lipase activity in adipose tissue has a counterregulatory role in the maintenance of body weight and adipose tissue mass in smokers. The higher the level of lipase activity when the weight-reducing influences of cigarettes cease, the greater the rate at which weight is gained during the first 3 weeks of abstinence. Cigarette smokers weigh less than nonsmokers and gain weight when they stop smoking. Increased activity of lipoprotein lipase in adipose tissue in some smokers may represent a compensatory response to reduced body weight. Consequently, it was hypothesized that the enzyme's activity may be related to the rate at which smokers gain weight when they stop smoking. To test this hypothesis, body weight and fasting lipoprotein lipase activity in adipose tissue was measured in 15 cigarette smokers before cessation of smoking. The changes in body weight during the first 2 weeks of abstinence were correlated with the baseline lipase activity ($r=0.82$, $p<0.0002$). This relationship remained significant in the 12 subjects who were still abstinent at 3 weeks ($r=0.63$, $p<0.03$). (Auth. Abs.)

84–1012. Carruthers, C.; Neilson, A. **Effect of 12-O-Tetradecanoyl-Phorbol-13-Acetate and 3-Methylcholanthrene on the Toxicity of Nicotine Applied to Mouse Skin.** *Oncology* 40(6): 404-409, November-December 1983.

The effect of the tumor promoter, 12-O-tetradecanoyl-phorbol-13-acetate (TPA) and 3-methylcholanthrene (MCA) on the toxicity of nicotine and the nicotine content in mouse skin was determined. TPA or MCA was applied to skin at time 0, nicotine was applied at various time intervals thereafter, and the nicotine—DNA ratio was determined 30 minutes later. After application of TPA, the nicotine—DNA ratio decreased from 120 at 1.8 hours to 80 at 18 hours, increased sharply from 24 hours, reached a maximum of 165 at 72 hours, and then decreased gradually. As the nicotine—DNA ratio increased, the mortality rate of the mice also increased, reaching a maximum of over 40 percent at 48 and 72 hours. When MCA was similarly applied, followed by nicotine, there was a decrease in the nicotine—DNA ratio similar to that induced by TPA and nicotine, followed by an average increase in the nicotine—DNA ratio of 130 at 72, 96, and 120 hours. The mortality rate was low. Finally, ethyl phenyl propiolate was applied at time 0, and nicotine at 24, 48, 72, 96, and 120 hours; the mortality rate was again low (nicotine was not determined in the skin). TPA induced some skin change 24, 48, 72, and 96 hours after application, which resulted in an increase in nicotine toxicity. Nicotine was not detected in the serum or plasma of mice treated with nicotine or TPA and nicotine, although acute toxicity reactions were observed, and the skin surface contained ample amounts of nicotine. (Auth. Abs. Mod.)

84–1013. Carta, P.; Cocco, P. L.; Lo Monaco, C.; Meloni, M.; Casula, D. **Carbossiemoglobina ed Abitudini al Fumo di Sigaretta. [Carboxyhemoglobin and the Habit of Cigarette Smoking.]** *L'Igiene Moderna* 78(6): 762-774, December 1982, Italian.

The relationship between cigarette smoking and carboxyhemoglobin (COHb) levels in venous blood was studied in 980 men. Mean levels of COHb were 0.97 percent in 245 nonsmokers, 1.26 percent in 192 ex-smokers, and 3.73 percent in 537 current smokers. Among smokers, COHb levels were dose-dependent; 3.06 percent in 215 light smokers (<10 cigarettes/day), 4.19 percent in 269 who smoked 11 to 20 cigarettes/day, and 4.64 percent in the 53 heavy smokers (>20 cigarettes/day). COHb levels also varied with the number of hours of smoking abstention and, in particular, with the amount smoked just before blood was drawn. Similar analyses were made for two occupational groups identified according to occupational exposure to carbon monoxide (CO), and similar response patterns were found. Differences in COHb levels between the high-CO exposure and the low-CO exposure occupational groups were not statistically significant. (Auth. Abs. Mod.)

84–1014. Clarke, P.B.S.; Kumar, R. **Characterization of the Locomotor Stimulant Action of Nicotine in Tolerant Rats.** *British Journal of Pharmacology* 80(3): 587-594, November 1983.

Tests of locomotor activity (photocell cages) were used to investigate the development of tolerance to nicotine in rats. Comparisons of (+)-nicotine (0.4-1.6 mg/kg s.c.) and (—)-nicotine (0.1-0.4 mg/kg s.c.) in tolerant rats showed that the (—)-isomer was at least 10 times more potent in stimulating motor activity. Subcutaneous pretreatment with mecamylamine (1.0 mg/kg) completely prevented the locomotor stimulant action of nicotine in tolerant rats, whereas chlorisondamine (0.01 or 0.1 mg/kg s.c.) only partially reduced it. When mecamylamine was given after an injection of nicotine, the locomotor stimulant action of nicotine was blocked, and nicotine actually reduced activity. A single intraventricular dose of chlorisondamine (2 µg) blocked the stimulant actions of nicotine for the duration of the experiment (23 to 24 days). Repeated exposure to the apparatus did not influence the rate at which tolerance was acquired. (Auth. Abs.)

84–1015. Damas, J. **Bref Apercu sur la Pharmacologie de la Nicotine. [Brief Summary of the Pharmacology of Nicotine.]** *Revue Medicale de Liege* 38(17): 641-643, September 1, 1983, French.

In tests on rats, nicotine was injected and immediately caused accelerated respiratory rhythm and convulsions. Without artificial respiration, an animal did not survive. Such experiments indicate that nicotine acts on the autonomous nervous system, the peripheral receptors, and the central nervous system. Some results show that nicotine may inhibit the formation of prostacyclins and increase platelet aggregation, and thus be a factor in thrombosis. Tolerance is developed to certain effects of nicotine, and although the first cigarette of the day produces cardiovascular effects, regular smokers do not suffer from nausea, vomiting, etc. Cessation of smoking may be accompanied by withdrawal symptoms that vary in severity depending on the person. It is also noted that (1) nicotine is highly water soluble and easily absorbed by the respiratory tract and mucous membranes of the mouth and nose; (2) in man, the half-life of nicotine is about 30 to 60 minutes; and (3) in lactating women, nicotine passes into the milk at concentrations as high as 0.5 mg/l.

84–1016. Eiden, L. E.; Eskay, R. L.; Scott, J.; Pollard, H.; Hotchkiss, A. J. **Primary Cultures of Bovine Chromaffin Cells Synthesize and Secrete Vasoactive Intestinal Polypeptide (VIP).** *Life Sciences* 33(8): 687-693, August 22, 1983.

The cholinergic agonists nicotine and carbachol stimulated the release of vasoactive intestinal polypeptide (VIP) and encephalin from bovine chromaffin cells in a dose- and calcium-dependent manner, although VIP could not be detected in extracts of bovine adrenal medulla or cortex. Immunoreactive VIP eluted from a reversed-phase chromatography column with the same retention time as authentic porcine VIP_{1-28}. Other neuroendocrine secretagogues that caused VIP and encephalin release were elevated potassium and veratridine. Both caused a dose-dependent release, the one with veratridine being calcium-dependent. Findings suggest that chromaffin cells of the bovine adrenal medulla in culture synthesize VIP_{1-28}, which is stored in secretory vesicles and released upon stimulation of cholinergic receptors on the surface of the cells.

84–1017. First, M. W. **Environmental Tobacco Smoke. 4.1. Exposure.** *European Journal of Respiratory Diseases* 65(Supplement 133): 137-139, 1984.

To understand the effects of environmental tobacco smoke (ETS), a firm linkage between exposure and retained dose must be established. The first priority should be exposure of the most sensitive individuals or those exposed to the highest levels of ETS, since useful information on effects is likely to be obtained most rapidly from these populations. While carbon monoxide (CO) has been used as an indicator of ETS, other substances can be used, depending on the specific effects under investigation, such as (1) nicotine for the acute effects of ETS; (2) total particulate matter fraction for lung function impairment studies; and (3) particulate matter for long-term chronic effects such as lung cancer. Questionnaires are a practical starting point for long-term studies, while personal sampling is as important as area monitoring. The problems in measuring the individual dose received from ETS are discussed, and important confounding factors in interpreting measurements are listed, as are specific research needs in the area of ETS exposure.

84–1018. Fung, H.-L.; Ruggirello, D.; Stone, J. A.; Parker, J. O. **Effects of Disease, Route of Administration, Cigarette Smoking, Food Intake on the Pharmacokinetics and Circulatory Effects of Isosorbide Dinitrate.** *Zeitschrift fur Kardiologie* 72(Supplement 3): 5-10, 1983.

The absorption and metabolism of oral isosorbide dinitrate (ISDN) were not significantly altered in the groups studied; however, the maximum percentage change in several of the circulatory parameters after ISDN was higher in nonsmokers compared with smokers. Smoking status did not affect the extent of ISDN absorption and clearance. Sublingual (5 mg) and oral (30 mg) ISDN were given under fasting conditions on two separate occasions to (1) eight patients with angina pectoris (AP); (2) nine patients with chronic congestive heart failure; (3) seven smoking AP patients; and (4) nine nonsmoking AP patients. Patients in group 1 were also given oral ISDN after a standard breakfast. Circulatory effects (i.e., heart rate, systolic and diastolic pressures) were measured over 3 hours after sublingual dosing and 6 hours after oral dosing in both the supine and standing positions. Plasma ISDN concentrations were measured by gas chromatography over the same periods. The systemic availability of oral ISDN was found to be about half that of sublingual ISDN, but the intersubject variability was large. Although food reduced the apparent absorption rate, the nonsignificant changes noted suggest that the standard dosage regimen of ISDN for these patient groups need not be altered. (Auth. Abs. Mod.)

84–1019. Hand, W. L.; King-Thompson, N. L.; Steinberg, T. H. **Interactions of Antibiotics and Phagocytes.** *Journal of Antimicrobial Chemotherapy* 12(Supplement C): 1-11, October 1983.

Certain factors, such as smoking, may influence antibiotic uptake by phagocytic cells and affect the efficacy of antibiotics in treatment of infection due to intracellular organisms. To establish those characteristics of drug and cell that mediate the antibiotic—phagocyte interaction, the uptake of radiolabeled antibiotics by rabbit alveolar macrophages (AM), human AM from smokers and nonsmokers, and human polymorphonuclear leukocytes (PMN) was studied. Relative entries of drug groups into the three types of phagocytic cells were similar. Human AM of smokers accumulated certain antibiotics more avidly than AM of nonsmokers. Rifampicin, clindamycin, and erythromycin achieved significantly higher levels in smokers than in nonsmokers after a 60 min incubation period. (Auth. Abs. Mod.)

84–1020. Harada, T.; Maita, K.; Nakashima, N.; Odanaka, Y.; Shirasu, Y. **Quantitative Studies of Biological Response in Hamsters Exposed to Tobacco Smoke and Effects of Vitamin C Supplement on Smoke Inhalation Toxicity.** *Japanese Journal of Veterinary Science* 45(5): 613-626, October 1983.

For quantitative evaluation of biological responses, male Syrian golden hamsters were exposed to smoke from 10, 20, or 30 cigarettes twice/day, 5 days/week, in a smoking machine. An additional group received dietary vitamin C supplement and was exposed to smoke from 30 cigarettes to study the effect of vitamin C on smoke inhalation toxicity. Animals were sacrificed after 4, 13, and 53 weeks of exposure. Smoke-exposed hamsters exhibited decreased body weight gain and food efficiency depending on dose and showed various tobacco-related histological changes in the respiratory tract. Histometrical evaluation revealed that smoke exposure enhanced alveolar macrophage mobilization and thickening of the laryngeal mucosa related to dose and duration of exposure. The vitamin C-supplemented group showed slightly improved body weight gain and food efficiency, significantly lower incidences of rhinitis, focal bronchial epithelial hyperplasia and bronchiolar adenomatoid lesion, and depressed alveolar macrophage mobilization compared with the smoke-exposed group at the same dose. Results indicate that alveolar macrophage count and thickness of the laryngeal mucosa may be useful in rating the biological damage elicited by cigarette smoke in hamsters. In addition, vitamin C may have some protective effect. (Auth. Abs. Mod.)

84–1021. Heraud, G.; Maizeray, B. **Oxycarbonisme Chronique des Fumeurs. [Chronic Carbon Monoxide Poisoning in Smokers.]** *Revue de Medecine* 24(31): 1441-1450, September 19, 1983, French.

Smoking, regardless of the brand used, results in chronic carbon monoxide poisoning. Tobacco smoke inhalation results in a carboxyhemoglobin level of 10 percent, reducing the oxygen transport capacity of the red blood cells by 10 percent also. Chronic carbon monoxide poisoning causes polycythemia in smokers, and affects the deformability of the red blood cell. It also appears to be very important in atherogenesis, as it disturbs the cholesterol/phospholipid balance in the atherogenic low density and high density lipoproteins. This would explain, to a large degree, the increasingly important role of tobacco in the process of atherogenesis. (Auth. Abs. Mod.)

84–1022. Hersey, P.; Prendergast, D.; Edwards, A. **Effects of Cigarette Smoking on the Immune System. Follow-Up Studies in Normal Subjects After Cessation of Smoking.** *Medical Journal of Australia* 2(9): 425-429, October 29, 1983.

Immune system effects were investigated in 35 subjects before, and 3 months after, cessation of smoking. Tests of natural killer cell (NK) activity against several target cells were made and immunoglobulin levels in serum and saliva were measured. Similar tests were conducted on 29 control subjects who continued to smoke. Results indicated a significant decrease in lymphocyte counts and a significant increase in NK activity against cultured melanoma cells in subjects who stopped smoking. Serum IgG and IgM levels rose significantly in those who ceased smoking cigarettes, but there was no change in IgA levels. Similar increases in immunoglobulin levels (IgA and IgG) in mucosal secretions (saliva) were noted after cessation of smoking. The NK activity and immunoglobulin levels of smokers who continued to smoke did not show significant changes. Results were consistent with the reversal of changes in immune function associated with smoking. (Auth. Abs. Mod.)

84–1023. Hosomi, J.; Nemoto, N.; Kuroki, T. **Genetic Differences in Metabolism of Benzo(*a*)pyrene in Cultured Epidermal and Dermal Cells of Responsive and Nonresponsive Mice.** *Cancer Research* 43(8): 3643-3648, August 1983.

Genetic differences in metabolism of benzo(*a*)pyrene (BaP) were investigated using cultured epidermal keratinocytes and dermal fibroblasts isolated from four inbred strains of mice: C3H/HeJms and C57BL/6J that are responsive, and DBA/2 and AKR/Jms that are nonresponsive in terms of inducibility of aryl hydrocarbon hydroxylase (AHH) activity. Primary cultures of epidermal and dermal cells isolated from newborn mice were treated with benz(*a*)anthracene for 24 hours. In both types of cells, AHH activity was induced in all four strains of mice, regardless of their responsiveness in vivo. In the epidermal cells, basal AHH activity varied within a relatively small range of 4.6 to 8.8 pmol/mg protein/hour, and the activity was increased 4.4 to 8.7 times by benz(*a*)anthracene treatment. There was no difference in the extent of the induction in different strains of mice. In dermal cells, the basal level of AHH activity was in the range of 3.6 to 9.1 pmol/mg protein/hour, and benz(*a*)anthracene treatment induced AHH, but to various degrees depending on the responsiveness in vivo. Epidermal cells of the responsive mice metabolized more than 90 percent of the added BaP in 48 hours, while those of nonresponsive mice metabolized only 60 to 70 percent. Dermal cells also metabolized BaP, but to a lesser extent than did epidermal cells, and there was no strain difference in its metabolism. Time-course studies revealed that epidermal cells of responsive mice metabolized BaP more rapidly than did those of nonresponsive mice. The activities of epidermal and dermal cells to metabolize BaP were further demonstrated by a cell-mediated mutation assay. Consistent with the results of time-course analysis of the metabolites, shorter treatments with BaP ($<$24 hours) showed a clear difference in BaP metabolism associated with responsiveness. (Auth. Abs.)

84–1024. Jarvis, M. J.; Russell, M.A.H. **Environmental Tobacco Smoke. 2.2. Measurement and Estimation of Smoke Dosage to Non-Smokers From Environmental Tobacco Smoke.** *European Journal of Respiratory Diseases* 65(Supplement 133): 68-75, 1984.

Data from a sample of 121 self-reported nonsmokers show that while overall mean levels of each marker measured were low, cotinine in plasma, saliva, and urine varied systematically with reported exposure to environmental tobacco smoke (ETS). Thus, biochemical markers, such as noninvasive samples of urine and saliva, can provide a sensitive guide to the recent daily life exposure to ETS. Carbon monoxide levels can be used to measure acute experimental exposure, but because of its short half-life, plasma nicotine is not suitable as an index of nicotine intake over several hours. It is noted that persons most heavily exposed to ETS are smokers exposed to their own mainstream and also sidestream smoke. Pipe and cigar smokers, most of whom never smoked cigarettes, would exhibit risks that would be an overestimation of the risks of ETS per se, since the group would include some who actively inhaled.

84–1025. Kasanof, D.; Kotch, A.; Little, D. N.; Neff, T. A. **The Vascular System. Could It Be Carbon Monoxide Poisoning?** *Patient Care* 17(19): 154-177, November 15, 1983.

Exposure to the byproducts of combustion (i.e., cigarette smoke, automobile exhaust, or campfire smoke), especially if exposure occurs within an enclosed space, near the floor, or at high altitudes, increases the likelihood of significantly elevated carboxyhemoglobin levels and carbon monoxide poisoning. Perhaps as many as 25 percent of cigarette smokers have increased red cell mass as compensation for elevated levels of carboxyhemoglobin. Persons who tend to steadily increase consumption and depth of inhalation (i.e., smokers of low-yield cigarettes) increase the exposure to carbon monoxide. In a smoke-filled room, nonsmokers can attain about a 5 percent level of carboxyhemoglobin. Passive smokers with cardiovascular disease experience approximately a 25 percent reduction in exercise tolerance; similar reductions have been observed in passive smokers with chronic obstructive pulmonary disease. Hazards of carbon monoxide exposure among smokers may also include higher accident/automobile accident rates (attributed to possible neuropsychiatric effects), and a wide range of cardiovascular and pulmonary diseases. Symptoms and diagnosis of carbon monoxide poisoning are described.

84–1026. Lafargue, P.; Faudon, R.; Bernard, P. **Tentative d'Approche Biologique de l'Oxygenation Tissulaire. Comparison des Resultats Obtenus Chez des Sujets Fumeurs et non Fumeurs. [Tentative Biological Approach to Oxygenation of the Tissue. Comparative Data for Smokers and Nonsmokers.]** *Medecine et Armees* 11(7): 611-613, October-November 1983, French.

In the framework of an antismoking campaign among French military personnel, hemoglobin (Hb), oxyhemoglobin (O_2Hb), carboxyhemoglobin (COHb), and 2,3-diphosphoglycerate (DPG) were determined in blood samples taken from 66 smokers of both sexes, aged 24 to 54 years. Samples were obtained before the start of a detoxification cure by acupuncture and 15 days after smoking cessation. Analytical data showed that COHb blood levels decreased significantly after treatment, indicating that the decrease was greater when cigarette consumption had been higher or the subjects had inhaled smoke; thus, observed COHb levels for ex-smokers of 40 cigarettes/day were 0.018, 0.056, and 0.104, when subjects never inhaled, inhaled occasionally, and inhaled systematically, respectively. O_2Hb levels increased as COHb levels decreased in the 49 subjects who had previously smoked 10 to 40 cigarettes/day, but decreased significantly in the subjects who had smoked over 40 cigarettes/day. Furthermore, the 2,3-DPG level decreased in all cases, although the decrease was not statistically significant. As expected, Hb blood level did not change significantly.

84–1027. Lehrer, S. B.; Barbandi, F.; Taylor, J. P.; Salvaggio, J. E. **Tobacco Smoke "Sensitivity"—Is There an Immunologic Basis?** *Journal of Allergy and Clinical Immunology* 73(2): 240-245, February 1984.

Results suggest that subjective tobacco smoke sensitivity is not caused by hypersensitivity to tobacco leaf or smoke antigens. Immunologic tests were conducted to determine if immediate skin reactivity and antibodies (reaginic and precipitating) to tobacco leaf/smoke antigens exist in man and whether or not these responses are related to subjective tobacco smoke sensitivity. Subjects (n=93), recruited on the basis of smoking history and/or claimed sensitivity to tobacco smoke, were skin-prick tested with tobacco smoke and leaf extracts, and sera were analyzed for reaginic and precipitating antibodies to these antigens. A significant number of the subjects had positive skin test and radioallergosorbent test (RAST) responses to tobacco leaf antigens, whereas only a small number responded to smoke antigens. RAST or skin test responses to leaf or smoke antigens did not correlate with symptoms of tobacco smoke sensitivity or smoking history, but did correlate with atopic status. Precipitins were detected only to tobacco leaf C (Kentucky air-cured burley tobacco compared with North Carolina flue-cured tobacco and Maryland tobacco) in 46 of the 93 persons, but did not correlate with smoking history or smoke sensitivity.

84–1028. Lilis, R.; Valciukas, J. A.; Weber, J.-P.; Fischbein, A.; Nicholson, W. J.; Campbell, C.; Malkin, J.; Selikoff, I. J. **Distribution of Blood Lead, Blood Cadmium, Urinary Cadmium, and Urinary Arsenic Levels in Employees of a Copper Smelter.** *Environmental Research* 33(1): 76-95, February 1984.

Exposure to cadmium-containing fumes and dusts and cigarette smoking contributed independently to cadmium (Cd) absorption and elevation of blood (Cd-B) and urinary (Cd-U) levels. A total of 776 copper smelter employees (680 active and 96 retirees and ex-employees) were examined for Cd-U, Cd-B, blood lead (Pb-B), zinc protoporphyrin (ZPP), and urinary arsenic (As-U). Another 144 men, never employed in the smelter, who had worked in copper mines (and sometimes in gold mines) were also examined. Mean Pb-B, ZPP, Cd-B, and As-U were significantly higher in active copper smelter employees than in retirees or miners, indicating exposure and absorption in the copper smelter. Significant correlations between Pb-B and Cd-B, and Cd-U and As-U were present, confirming the common source of absorption. A marked effect of smoking on Cd-B was present: regardless of employment category, smokers showed significantly higher Cd-B levels compared with nonsmokers. Age was also a factor, since the highest Cd-U levels were found in retirees who smoked. Nevertheless, for all smoking categories, Cd-B levels were significantly higher in active employees, indicating the independent contribution of exposure to cadmium in the smelter. (Auth. Abs. Mod.)

84–1029. Lynch, C. J. **Environmental Tobacco Smoke. 2.1. Half-Lives of Selected Tobacco Smoke Exposure Markers.** *European Journal of Respiratory Diseases* 65(Supplement 133): 63-67, 1984.

Analysis of blood cotinine and expired carbon monoxide (CO) in 107 subjects who abstained from smoking for 24 hours revealed overall half-lives of 11.3 hours for cotinine (range 7.1 to 29.6) and 11.3 hours for CO (range 7.9 to 19.3) in the 55 men, and 15.2 hours for cotinine and 11.6 hours for CO (range 6.0 to 20.5) among the 52 females. The cotinine half-life was much shorter, while the CO half-life was much longer than expected. Almost no difference in cotinine and CO values was observed between two subject groups, one composed of smokers (average consumption 35.2 cigarettes/day, baseline cotinine 281 ng/ml), the other of participants in a smoking cessation program (average consumption 28.1 cigarettes/day, baseline cotinine 418 ng/ml). Stratification by gender also revealed no significant differences between groups. More detailed data are given for the males: average consumption, 34.2 cigarettes/day (range 20 to 70); average baseline cotinine level 301 ng/ml (range 132 to 775); and average expired CO, 42.5 ppm (range 26 to 73). It is suggested that previously reported half-lives should be re-examined.

84–1030. Mannisto, J. **The Effects of Cigarette Smoke on the Fate of Arachidonic Acid in Rat and Hamster Lungs.** *Prostaglandins* 26(5): 681-688, November 1983.

In a study of the effects of cigarette smoke on the fate of exogenous arachidonic acid (AA) in isolated perfused rat and hamster lungs, pulmonary metabolism of AA was changed by cigarette smoke ventilation but not by preexposure of animals. Both incorporation of AA into lung lipids and efflux of metabolites from the lungs were affected by cigarette smoke. ^{14}C-arachidonate was infused into the pulmonary circulation and the metabolites in the effluent and the distribution of radiolabel in lung lipids were analyzed. Preexposure of animals to cigarette smoke daily for 3 weeks did not change the amounts of metabolites in lung effluent or the incorporation of AA into lung lipids. Cigarette smoke ventilation of lungs from preexposed hamsters changed the amounts of some metabolites of AA in the effluent. The distribution of radioactivity in the lung tissue was changed during smoke ventilation in both rat and hamster. The amount of radioactivity in some lipid fractions was increased by cigarette smoke, the increase being greatest in triacylglycerols. (Auth. Abs. Mod.)

84–1031. Paoletti, R. **Wechselwirkungen Zwischen Thrombozyten und Gefasswand bei Hypercholesterinamie.** [Interaction Between Thrombocytes and Vascular Wall in Hypercholesterolemia.] *Medizinische Klinik* 78(8): 256-259, April 29, 1983, German.

Hyperlipidemia disturbs the equilibrium between the formation of prostacyclin and thromboxane, and nicotine from cigarettes acts as a specific inhibitor of prostacyclin biosynthesis. Thus, the prostacyclin—thromboxane balance is disturbed in favor of thromboxane in smokers, as has been noted in clinical reports of hyperaggregation and autoaggregation in heavy smokers. With hyperlipidemia, prostacyclin formation is reduced, and smoking further aggravates the condition, which explains epidemiologic findings of exponential risk when both risk factors are present.

84–1032. Queneau, P.; Ollagnier, M.; Decousus, H.; Perpoint, B.; Brazier, J. L.; Faucon, L. **Influence du Tabac sur les Taux**

Seriques et la Pharmacocinetique de la Phenylbutazone et de l'Oxyphenbutazone. [Influence of Tobacco on the Serum Levels and the Pharmacokinetics of Phenylbutazone and Oxyphenbutazone.] *Revue du Rhumatisme et des Maladies Osteo-Articulaires* 50(4): 293-297, April 1983, French.

To determine whether serum levels of phenylbutazone (PB) were reduced by smoking, changes in serum levels of PB and oxyphenbutazone (OPB), the hydroxylated metabolite of phenylbutazone, were compared during 10 days of administration, in nine smokers and six nonsmokers. Blood samples were taken daily at 8:00 a.m., and PB and OPB were assayed by high performance liquid chromatography. Comparison of mean serum levels of PB in smokers and nonsmokers revealed a statistically significant reduction ($p \leq 0.05$) in smokers. OPB was significantly reduced in smokers on days 9 and 10 only. It appears that tobacco smoke accelerates the metabolism of phenylbutazone and oxyphenbutazone by a mechanism of enzyme induction. It is proposed that the therapeutic activity of phenylbutazone is reduced in smokers, but this has not been confirmed by a reliable clinical evaluation. (Auth. Abs. Mod.)

84–1033. Radojicic, B.; Lekic, T.; Koracevic, D.; Pavlovic, M.; Vagaja, M. **Delovanje Organofosfornih Insekticida i Nikotina na Aktivnost Enzima Katalaze. [Effect of Organophosphoric Insecticides and Nicotine on the Activity of Catalase Enzymes.]** *Acta Medica Medianae* 21(6): 5-10, April 1982, Serbo-Croatian.

Results of a Yugoslav study indicate that workers exposed to both organophosphate insecticides and nicotine had significantly increased urine catalase levels. Three groups were studied: (1) 128 men exposed to organophosphate insecticides (parathion, malathion, etc.); (2) 143 men exposed to such insecticides and nicotine; and (3) 105 nonexposed controls. Urine catalase levels were 8.48 percent higher in the first group and 6.24 percent higher in the second, compared with controls. Urine nicotine levels in the exposed group were more than 1,000 times higher than levels in controls. It is noted that increased catalase activity following toxic exposure to organophosphate insecticides and nicotine may damage blood-forming organs as well as catalase-containing tissues and cells, and that the probability of such changes increases in occupationally exposed smokers.

84–1034. Savage, A. O.; Sanya, I. O. **Effect of Ketamine on Cholinergic Transmission in the Isolated Guinea-Pig Ileum Preparation.** *Archives Internationales de Pharmacodynamie et de Therapie* 263(1): 103-112, May 1983.

Low concentrations of ketamine ($<4.2\ \mu M$) applied to isolated guinea pig ileum produced slight increases in nicotine-, carbachol-, and electrically-induced contractions. Higher concentrations caused concentration-dependent inhibition of the contractions produced by all three interventions. Ketamine inhibition of nicotine-induced contractions was found to be relatively independent of external calcium ion concentration. Apparently nicotine, by stimulating ganglion cells, caused the release of the cholinergic transmitter, acetylcholine, whose output was increased by low, and blocked by high, concentrations of ketamine. Results obtained suggest that low concentrations of ketamine facilitate, while higher concentrations inhibit cholinergic transmission by an action exerted on postganglionic parasympathetic nerve terminals, although effects of the compound on parasympathetic ganglia cannot be excluded. (Auth. Abs. Mod.)

84–1035. Sepkovic, D. W.; Haley, N. J.; Wynder, E. L. **Thyroid Activity in Cigarette Smokers.** *Archives of Internal Medicine* 144(3): 501-503, March 1984.

Substantial decreases in serum thyroxine (T_4) and triiodothyronine (T_3) concentrations were found in heavy smokers compared with nonsmoking controls in an investigation of the relationship between cigarette smoking and thyroid function. Volunteers (99 males, 101 females) were divided into four groups according to self-reported and biochemical data, including blood carboxyhemoglobin, plasma cotinine, and thiocyanate, and were classified as nonsmokers ($n = 50$, controls), light smokers ($n = 50$), medium smokers (50), and heavy smokers (50). Self-reported smoking behavior correlated closely with biochemical measurements. Plasma T_3 and T_4 concentrations in heavy smokers were significantly below values for nonsmokers, and light and moderate smokers demonstrated a slight decrease in thyroid hormone levels, implying a dose—response relationship between increased absorption of cigarette smoke thiocyanate and decreased thyroid hormone levels. Plasma measurement of thyroid-binding globulin capacity found no differences between smokers and nonsmokers. Reduced serum thyroid hormone levels were not accompanied by a substantial change in serum thyrotropin concentrations. The free T_4 index, free T_3 index, and T_4 to T_3 ratio were not substantially different for heavy smokers and nonsmokers. (Auth. Abs. Mod.)

84–1036. Sloan, J. W.; Martin, W. R.; Todd, G. D. **Multiple Nicotine Binding Sites in Rat Brain P_2 Fraction.** *Psychopharmacology Bulletin* 19(3): 402-408, 1983.

Data indicate that, in the rat brain P_2 preparation, there are at least two and possibly three nicotine binding sites and a cooperativity site that may or may not be distinct. The following evidence suggests that positive cooperativity occurs at the binding site: (1) the configuration of Scatchard plots derived from both ($\pm$)-(^{3}H)-nicotine binding and (—)-nicotine inhibition of ($\pm$)-(^{3}H)-nicotine binding; (2) coefficients >1.0 for nicotine binding at low concentrations; (3) clear enhancement of ($\pm$)-(^{3}H)-nicotine binding by low concentrations of carbachol; and (4) enhancement of nicotine binding by anabasine, lobeline, (+)-nicotine, and mecamylamine, but not by cytisine, cotinine, and atropine, indicating selectivity among cholinergic drugs for enhancing ($\pm$)-(^{3}H)-nicotine binding. Results support the hypothesis that ($\pm$)-(^{3}H)-nicotine binds to multiple sites in the rat brain P_2 preparations, but with a complexity not found before.

84–1037. Smith, R. B.; Gwilt, P. R.; Wright, C. E., III. **Single- and Multiple-Dose Pharmacokinetics of Oral Alprazolam in Healthy Smoking and Nonsmoking Men.** *Clinical Pharmacy* 2(2): 139-143, March-April 1983.

Although not significantly different, alprazolam elimination was more rapid in smokers than in nonsmokers. Ten healthy men (five smokers and five nonsmokers), aged 19 to 42 years, received a single oral 1 mg dose of alprazolam (a new benzodiazepine) followed 48 hours later by 0.5 mg alprazolam every 8 hours for 6 days. Mean plasma alprazolam concentrations during the single-dose phase were comparable between groups. However, during the multiple-dose phase, mean plasma concentrations were 15 to 30 percent lower in smokers. Total body clearance of alprazolam was 24 percent greater and the apparent volume of distribution was 17 percent less in smokers. The elimination half-life was 49 percent greater in nonsmokers. These differences were not significant. Changes were not observed in elimination half-life or clearance after alprazolam cessation. Steady-state pharmacokinetic values during the multiple-dose phase correlated with values observed following the single dose. (Auth. Abs. Mod.)

84–1038. Sorsa, M.; Falck, K.; Maki-Paakkanen, J.; Vainio, H. **Genotoxic Hazards in the Rubber Industry.** *Scandinavian Journal of Work, Environment and Health* 9(2, Special issue): 103-107, April 1983.

Smoking was clearly associated with significantly increased numbers of sister chromatid exchanges (SCE) and chromosome aberrations among 55 rubber workers (31 smokers) and 35 controls (14 smokers). Biological monitoring methods were used to identify job categories with possible exposure to potentially genotoxic chemicals. Urine samples, collected and analyzed for mutagenic activity by the bacterial fluctuation test, showed the highest individual mutagenic activities among workers in the weighing and mixing departments, and some high individual values among vulcanizers. SCE and structural chromosome aberrations were also analyzed from samples of peripheral blood. Among the workers in different job categories, the highest SCE frequencies were found for mixers who smoked. Also, nonsmoking cleaners of the mixing department differed significantly from the nonsmoking controls in this respect. However, only the group of nonsmoking weighers showed an increase in the number of chromosome aberrations ($p < 0.01$) when compared with controls. Results suggest the need for improved occupational hygiene in the weighing and mixing departments of rubber plants so that exposure to potentially hazardous genotoxic chemicals can be decreased. (Auth. Abs. Mod.)

84–1039. Sram, R. J.; Samkova, I.; Hola, N. **High-Dose Ascorbic Acid Prophylaxis in Workers Occupationally Exposed to Halogenated Ethers.** *Journal of Hygiene, Epidemiology, Microbiology, and Immunology* 27(3): 305-318, 1983.

The influence of smoking habits was considered as part of a larger study of the possible prophylactic effect of ascorbic acid (AA) in 77 workers (42 smokers, 35 nonsmokers) occupationally exposed to carcinogens (halogenated ethers). The effect of prophylactic treatment was assessed by cytogenetic analysis of chromosome aberrations and by determining blastic transformation and mitotic index in peripheral lymphocytes. Blood samples were taken at the beginning and end of the 5-month treatment period. Differences between smokers and nonsmokers exposed to halogenated ethers were nonsignificant, nor were there significant differences between synthetic-resin-positive controls and nonexposed controls. Findings did not confirm observations on the relationship between smoking and frequency of aberrant cells, but results of AA prophylaxis in a subgroup of halogenated ether-exposed workers with at least 4 percent aberrant cells were more favorable in nonsmokers than in smokers. Overall, evidence suggests that long-term prophylactic administration of AA to subjects exposed to bis (chloromethyl) ether or chloromethyl methyl ether has a distinctly positive effect.

84–1040. Takayanagi, I.; Kizawa, Y. **A Possible Site of Action of Nicotine in the Bronchial Smooth Muscle Preparation of Guinea-Pig.** *Journal of Pharmacy and Pharmacology* 35(10): 682-683, October 1983.

Nicotine (10^{-6} to 10^{-3} M) dose-dependently produced contractions in guinea pig bronchial smooth muscle, with a maximum response of 38.5 ± 4.0 percent of that of acetylcholine. No inhibitory response to nicotine was observed in any of the preparations, and all were relaxed by papaverine (10^{-4}). Hexamethonium and pentolinium reduced the contractile response to nicotine, a reduction not affected by atropine. Indomethacin, tetrodotoxin, or the acetylcholine release inhibitor SX-284, did not influence the nicotine-induced contractions. Field stimulation induced a contractile response equal to about 40 percent of the nicotine response. Physostigmine treatment potentiated the field-induced contractions, but did not affect nicotine-induced responses. Separate experiments showed that tissue concentration of cyclic GMP was significantly increased by acetylcholine but not by nicotine. Results suggest that nicotine does not act via stimulation of cholinergic ganglion cells, and that the nicotine site of action is on smooth muscle cells and not nerve cells.

84–1041. Totti, N., III.; McCusker, K. T.; Campbell, E. J.; Griffin, G. L.; Senior, R. M. **Nicotine Is Chemotactic for Neutrophils and Enhances Neutrophil Responsiveness to Chemotactic Peptides.** *Science* 223(4632): 169-171, January 13, 1984.

Nicotine was found to be chemotactic for human neutrophils (which contribute to chronic bronchitis and pulmonary emphysema associated with cigarette smoking), but not monocytes. Unlike many factors chemotactic for polymorphonuclear neutrophils (PMNs), nicotine did not stimulate the release of superoxide anion or cause granulation. Nicotine produced a concentration-dependent migratory response of PMNs obtained from nonsmokers, maximal at 30.8 μM. Since nicotine was found to enhance PMN responsiveness to other chemotactic factors, it may thus alter receptor affinity for other chemoattractants or affect membrane fluidity, causing a faster startup response to chemoattractants. While the optimum concentration for nicotine-induced PMN chemotaxis is about 100 times higher than typically found in plasma of cigarette smokers, during smoking the intrapulmonary concentration may be much higher than in plasma. It is suggested that nicotine, by promoting pulmonary inflammation, may be a major smoke component in the pathogenesis of lung injury in cigarette smokers.

84–1042. Vehovszky, A.; Salanki, J. **Pharmacological Characterization of Postsynaptic Potentials Evoked in the Bimodal Pacemaker Neuron of *Helix Pomatia* L.** *Acta Physiologica Hungarica* 62(1): 35-46, 1983.

Evoked excitatory postsynaptic potentials (EPSP) on the identified RPal neuron of *Helix pomatia* L. were reduced or blocked by nicotine, atropine, and d-tubocurarine. The two components of inhibitory postsynaptic potential (IPSP) were different in pharmacological sensitivity: (1) slow IPSP was partly or totally eliminated by ergometrine and chlorpromazine, and reduced by atropine, nicotine, and propranolol; while (2) fast IPSP was reduced only in the presence of ergometrine and could not be blocked by either of the applied drugs. In the presence of nicotine, spikes were not evoked by stimulation. Although EPSPs occurred, both the number and amplitude were reduced. Fast IPSP became more visible, partly because the slow IPSP decreased both in amplitude and duration. The effect of nicotine could not be totally washed out. In the presence of nicotine, the endogenous pattern of the cell also changed; the amplitude of the interburst hyperpolarization was reduced significantly, frequency of the spikes decreased, and later the generation of action potentials ceased. This effect could not be reversed by washing. Participation of cholinergic transmission seems to be essential in the evoked EPSP, but its partial involvement in the slow IPSP can also be supposed. A dopaminergic mechanism may take part in the generation of both components of IPSP, but the receptors responsible for the slow IPSP were sensitive to other catecholamine antagonists as well, referring to a more complex origin or to the involvement of an unknown transmitter. Comparison of postsynaptic potentials evoked by stimulation of different nerves shows that presynaptic areas belonging to various peripheral sources are overlapped on the RPal neuron, and probably act by similar transmitter substances. (Auth. Abs. Mod.)

84–1043. Verhoeff, A. P.; Van der Velde, H.C.M.; Boleij, J.S.M.; Lebret, E.; Brunekreef, B. **Detecting Indoor CO Exposure by Measuring CO in Exhaled Breath.** *International Archives of Occupational and Environmental Health* 53(2): 167-173, 1983.

Only among smokers did exhaled carbon monoxide (CO) reach significantly increased levels in residents of Dutch flats with CO-producing water heaters. CO levels in exhaled breath were measured in 29 residents of flats equipped with a flueless geyser (an instantaneous gas-fired water heater). The selected flats had a geyser with a CO concentration of more than 250 ppm in its flue gases. Small, but in some cases statistically significant increases in CO levels in exhaled breath were found in both smokers and nonsmokers, and after periods of cooking and dishwashing when the geysers had been used. Calculated carboxyhemoglobin levels remained well below 2.5 percent for nonsmokers, but were generally higher for smokers, reaching 3.89 percent in some cases. (Auth. Abs. Mod.)

84–1044. Wakade, A. R.; Wakade, T. D. **Contribution of Nicotinic and Muscarinic Receptors in the Secretion of Catecholamines Evoked by Endogenous and Exogenous Acetylcholine.** *Neuroscience* 10(3): 973-978, 1983.

Using nicotine and muscarine as agonists, and hexamethonium and atropine as antagonists, it was determined that the rat adrenal gland contains nicotinic and muscarinic receptors, both linked to catecholamine secretion. These receptors, however, are activated in a different manner by endogenous and exogenous acetylcholine. The ratios of epinephrine to norepinephrine in the perfusate were comparable before and after stimulation of the adrenal gland with nicotine, acetylcholine, or potassium ions. Tetrodotoxin blocked the nicotine-evoked secretion by about 75 percent, but had very little effect on secretion evoked by muscarine. It is concluded that nicotinic receptors are primarily concentrated in the synaptic zones and are involved in the physiological stimulation of the adrenal medulla, whereas extrasynaptic regions contain a mixture of nicotinic and muscarinic receptors activated by injected acetylcholine. The tetrodotoxin data indicate that physiologically released acetylcholine can generate action potentials through synaptic nicotinic receptors to initiate an increase in calcium influx and subsequent secretion of catecholamines. (Auth. Abs. Mod.)

84–1045. Watanabe, T.; Koizumi, A.; Fujita, H.; Kumai, M.; Ikeda, M. **Cadmium Levels in the Blood of Inhabitants of Nonpolluted Areas in Japan With Special References to Aging and Smoking.** *Environmental Research* 31(2): 472-483, August 1983.

While the effects of passive smoking could not be confirmed, smoking had a dose-dependent effect on blood cadmium levels, which leveled off when consumption was ≥ 30 cigarettes/day. Blood samples (2,259, 826 males and 1,433 females), collected in winter from inhabitants (primarily farmers) in areas with no known manmade pollution, were analyzed for cadmium. Levels were lower among young adults and increased gradually, reaching a plateau in those aged 40 to 59. Values in females were significantly higher than in males. The sex difference was positive ($p<0.01$) even when levels in 77 husband and wife pairs (all nonsmokers aged 40 to 59) were compared. Seasonal variation of cadmium levels, checked in 523 separate samples, appeared negligible, and variation by geographic location was of doubtful significance. The estimated ratio of cadmium doses by two routes (gastrointestinal tract and lungs) was in agreement with the ratio of the blood cadmium level among nonsmokers and the additional increase in the level due to smoking. (Auth. Abs. Mod.)

See also **84–1114**

MORTALITY AND MORBIDITY

84–1046. Berkel, J.; De Waard, F. **Mortality Pattern and Life Expectancy of Seventh-Day Adventists in the Netherlands.** *International Journal of Epidemiology* 12(4): 455-459, December 1983.

Analysis of health practices and mortality patterns among Seventh-Day Adventists (SDA) indicates the low mortality from ischemic heart diseases and certain cancers among SDAs is related to abstinence from cigarette smoking, while an appropriate diet seems to improve colon cancer mortality rates. While 98.4 percent of SDAs are nonsmokers, compared with 52.8 percent of the general Dutch population, among the few who smoke, none consume more than 10 cigarettes/day, compared with 27.7 percent of the smokers in the Dutch population. Of a total of 522 deaths among SDAs during the 1968 to 1977 period, causes could be ascertained in 482. The standard mortality ratio (SMR) was 0.45 for total mortality, 0.50 for cancer, and 0.41 for cardiovascular diseases. Mean age of death was 75.3 years for males and 77.4 years for females, significantly higher than age of death of the general Dutch population.

84–1047. Damiani, P.; Masse, H. **Mortalite par Cause et Tabac: Application d'un Modele de Liaison et Evaluation de la Consommation de Tabac par Sexe et par Age. [Mortality by Cause and Smoking: Application of a Regression Model and Evaluation of Tobacco Consumption by Sex and Age.]** *Journal de la Societe de Statistique de Paris* 121(2): 81-89, 1980, French.

Relative per capita tobacco consumption in France by sex and age was evaluated, using 1969 statistics. Based on simple hypotheses and regional mortality data for cause by sex and age, mathematical regression models were derived, making it possible to calculate regression coefficients between the logarithm of mortality rate and cumulative consumption of tobacco. The models show that the coefficient for respiratory tract cancer is constant, but decreases exponentially with age for all other diseases. The correlation coefficients of the simple regressions were found to be positive for nearly all causes and were the highest for cancers of the respiratory tract, the urinary and genital organs, and liver cirrhosis among men aged 45 to 64 years. The coefficients for women were 1.6 to 8.2 times higher than for men. The models are applied to measure relative mortality from a given cause versus tobacco consumption. A sample calculation shows that for 45- to 64-year-old men, an increase in consumption of one cigarette/day would increase relative respiratory cancer mortality by 13.4 percent.

84–1048. Gillis, C. R.; Hole, D. J.; Hawthorne, V. M.; Boyle, P. **Environmental Tobacco Smoke. 3.6. The Effect of Environmental Tobacco Smoke in Two Urban Communities in the West of Scotland.** *European Journal of Respiratory Diseases* 65(Supplement 133): 121-126, 1984.

Environmental tobacco smoke (ETS) exposure, smoking, and mortality rates were analyzed for 8,128 subjects who attended a multiphasic screening unit between 1972 and 1976. At screening, subjects were between 45 and 64 years of age, and 97.6 percent were male/female partnerships. The prevalence of infected spit, persistent spit, dyspnea, and hypersecretion was slightly higher in ETS-exposed nonsmokers than in controls (nonexposed nonsmokers). Among ETS-exposed females, angina and ECG abnormalities were slightly more common than in controls, while reverse trend was seen among males. Analysis of male lung cancer mortality, however, showed rates of 4/10,000 in controls, 13/10,000 for ETS-exposed nonsmokers, 22/10,000 in smokers, and 24/10,000 in ETS-exposed smokers. No such trend was noted among the women. Overall myocardial infarction mortality was slightly higher in the ETS-exposed group, but no other differences in smoking-related mortality were found.

84–1049. Lennes, G. **Statistiques Relatives Aux Problemes du Tabagisme. [Relevant Statistics to the Problem of Smoking.]** *Revue Medicale de Liege* 38(17): 643-647, September 1, 1983, French.

Results are summarized from four large prospective studies conducted with British doctors, United States military veterans, Canadian military veterans, and a sample of American males. Excess total mortality associated with tobacco use was 1.28, 1.84, 1.65, and 1.83 for cigarette use only; 1.19, 1.51, 1.23, and 1.54 for cigarettes plus other forms of tobacco; and 1.01 for cigar use (only the British study). Excess mortality among pipe smokers was reported as 1.07 in American veterans, 1.10 in Canadian veterans, and 0.86 in American males. Mortality rates were given as a function of the age of smoking onset for the two American studies. In general, mortality increased with

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the number of cigarettes smoked, regardless of age of smoking onset. Mortality rates in ex-smokers were included as a function of quit time compared with nonsmokers and current smokers. Among subjects who reported low consumption rates and had quit for 10 years or more, mortality rates were about the same as for nonsmokers, while rates were considerably higher for quitters who had high levels of previous tobacco consumption. Statistical analytic techniques and uses of prospective and retrospective epidemiological data are discussed.

84–1050. Schoenbach, V. J.; Wagner, E. H.; Karon, J. M. **The Use of Epidemiologic Data for Personal Risk Assessment in Health Hazard/Health Risk Appraisal Programs.** *Journal of Chronic Diseases* 36(9): 625-638, 1983.

Risk projections for a 41-year-old white male who smokes a pack/day are 1.9 for lung cancer and 1.5 for arteriosclerotic heart disease. Health hazard/health risk appraisal programs (HHA/HRA) employ personal risk assessment as an educational and motivational technique to encourage the adoption of healthier lifestyles by health education clients. In reviewing the scientific basis of HHA/HRA assessments, it is pointed out that the 1.5 risk factor reflects the fact that many 40- to 45-year-old white males smoke. A nonsmoker would be assigned a risk factor of 0.4, less than half the average risk. Thus the implied epidemiologic risk ratio for smoking two packs/day (vs. not smoking) is 3.75. There are severe limitations in both the data and the risk estimation procedures. Various undocumented assumptions enter into the projections, and several key aspects are arbitrary. Proposals for improvement of the procedure generally require unavailable data. Attention has largely focused on increasing mathematical sophistication (e.g., adjustment for competing risks) while ignoring problems in accuracy of client data from which projections are calculated and serious questions about the health education messages implicit in the appraisal results. HHA/HRA programs should be candid about the limitations of the technique. Use of appraisal results for evaluation purposes is questionable. The health education messages produced by such programs deserve attention, since their appropriateness, especially for clients who are not white, middle class, and middle-aged, has not been established. (Auth. Abs. Mod.)

84–1051. Sterling, T. D. **Perceived vs. Actual Risks: The Problem of Multiple Confounding.** In: Covello, V. T.; Flamm, W. G.; Rodricks, J. V.; Tardiff, R. G. (Editors). *The Analysis of Actual Versus Perceived Risks.* Advances in Risk Analysis, Volume 1, New York, Plenum Press, 1983, pp. 203-212.

Failure to consider occupational exposure in evaluating smoking as a risk factor causes misleading conclusions to be drawn. Blue-collar workers are more apt to (1) be smokers, (2) smoke unfiltered cigarettes, (3) have more children, and (4) be exposed to dust and toxic fumes (as are their families) than white-collar workers. Occupation is a major determinant of socioeconomic class and nutritional factors. If wives of smokers are shown to have more respiratory disorders than nonsmokers' wives and occupation is not considered, or if the incidence of respiratory disorders among children is higher in households where the father smokes, it may reflect either occupational exposure or that there are more children in blue-collar families. The assessment of smoking risk changes radically when placed in the context of variables confounded with smoking (occupation, education, age, and socioeconomic status). Recent studies reporting higher risks of lung cancer for smokers' wives compared with nonsmokers' wives in Japan failed to consider kerosene stove emissions as well as class and income differences in smoking rates in Japan. Mortality statistics on smoking also fail to consider occupation: since nonsmokers tend to be older than smokers, the expected age at death of nonsmokers is bound to be higher than that of smokers. Social, industrial, and personal factors contributing to risk must be part of any risk assessment.

NEOPLASTIC DISEASES

84–1052. Ames, R. G.; Amandus, H.; Attfield, M.; Green, F. Y.; Vallyathan, V. **Does Coal Mine Dust Present a Risk for Lung Cancer? A Case—Control Study of U.S. Coal Miners.** *Archives of Environmental Health* 38(6): 331-333, November-December 1983.

Results of two case—control studies, based on 317 white male fatal lung cancer cases, found the expected increased risk of lung cancer in cigarette smokers. The studies evaluated the relationship between the risk of lung cancer mortality and coal mine dust exposure, controlling for smoking status. The one-to-one matched-case design allowed examination of dust exposure and smoking, while the two-to-one matched-case design was used to examine the lung cancer risk of coal dust exposure independent of cigarette smoking. No evidence of a coal mine dust exposure—lung cancer risk was found. There also was no evidence of an interactive effect between cigarette smoking and coal mine dust exposure. (Auth. Abs. Mod.)

84–1053. Ames, R. G.; Gamble, J. F. **Lung Cancer, Stomach Cancer, and Smoking Status Among Coal Miners: A Preliminary Test of a Hypothesis.** *Scandinavian Journal of Work, Environment and Health* 9(5): 443-448, October 1983.

A tentative test of the Meyer hypothesis, using coal miner mortality records, is described. The hypothesis suggests that the carcinogens in smoke are a risk for stomach cancer in persons with nonimpaired lung clearance and a risk for lung cancer in persons with impaired clearance. Based upon a case-referent study comparing 46 white male coal miners who died from stomach cancer in the United States with age-matched miners who died from lung cancer and with reference miners who died from other cancers or from noncancer, nonaccident causes, the present study failed to confirm the Meyer hypothesis. The data suggest a reverse relationship, namely, that airway obstruction may be a precondition for stomach cancer and normal pulmonary function a precondition for lung cancer. Different dust agents were found to be involved with cancers at the different sites. For coal miners with airway obstruction, greater years of coal mine dust exposure were found to pose a slightly elevated stomach cancer risk (odds ratio 3.64, not significant), while for miners with normal ventilatory function, cigarette smoking posed a disproportionately elevated lung cancer risk (odds ratio 7.00, not significant). (Auth. Abs. Mod.) See also 84–1061.

84–1054. Benhamou, S.; Hill, C. **Epidemiologie du Cancer Bronchique. [The Epidemiology of Lung Cancer.]** *La Revue du Praticien* 33(25): 1279-1280, 1285-1286, 1289-1291, May 1, 1983, French.

Tobacco is the main risk factor for lung cancer in France, accounting for 90 percent of all cases. Among French males, lung cancer mortality tripled between 1952 and 1977. The age-standardized rate rose from 15/100,000 in 1952 to 48/100,000 in 1977. Among females, however, the lung cancer mortality rate has remained steady: 5/100,000 in 1952 and 6/100,000 in 1977. Of other known risk factors, the most important are asbestos and radon. Although other substances are suspected of involvement in lung cancer etiology, the required epidemiological studies are difficult to undertake because of the confounding effects of smoking. (Auth. Abs. Mod.)

84–1055. Breeden, J. H. **Alcohol, Alcoholism, and Cancer.** *Medical Clinics of North America* 68(1): 163-177, January 1984.

Alcohol appears to act synergistically with tobacco in the etiology of cancers of the upper aerodigestive tract. Alcohol itself is not a primary carcinogen; however, alcohol may act as a cocarcinogen by solubilizing, activating, or catabolizing true carcinogens from tobacco smoke or other sources. Prospective studies of heavy drinkers show an overall excess of about 70 percent for cancer mortality. Even so, high alcohol consumption is strongly associated with heavy smoking, and the majority of these excess deaths are related to tobacco use. For oropharyngeal cancers, it is estimated that 33 percent are attributable to smoking and 43 percent to alcohol. The relative risk for head and neck neoplasms associated with alcohol is about 1.1 to 7.0 for low tobacco use groups and 5.0 to 20.0 for high tobacco use groups. The specific role of alcohol in carcinogenesis, the epidemiology of cancer of various sites relative to the type of alcoholic beverage consumed, and treatment-related aspects of the alcoholic cancer patient are discussed.

84–1056. Burch, P.R.J. **The Surgeon General's "Epidemiologic Criteria for Causality." A Critique.** *Journal of Chronic Diseases* 36(12): 821-836, 1983.

The methodology of the 1982 Report of the Surgeon General (U.S. Department of Health and Human Services, Public Health Service, Office of Smoking and Health, DHHS Pub. No. 82-50179, 1982, 341 pp.) is examined with special reference to smoking and lung cancer. It is shown that, plausibly interpreted, the five criteria for causality (described in Part II of the report), which have guided the judgment of committees since 1964, are not satisfied by the epidemiologic evidence for lung cancer. An underlying weakness in all the Reports is failure to recognize all the logical possibilities inherent in an association between smoking and a disease. The five criteria and the subjective method of "judgment" are inappropriate to a scientific analysis, and should be replaced by the objective testing of hypotheses. Limitations in the evidence and in concepts about tobacco carcinogenesis preclude definitive conclusions. Nevertheless, the entire association between cigarette smoking and lung cancer (at least in male Caucasoid populations) is unlikely to be explained by causation. (Auth. Abs. Mod.) See also 82-1299 and 84-1071.

84–1057. Burton, R. C. **Smoking, Immunity, and Cancer.** *Medical Journal of Australia* 2(9): 411-412, October 29, 1983.

Smoking produces a stable alteration in circulating T lymphocyte subsets, and is also immunosuppressive of B lymphocytes and natural killer (NK) cell activity. Studies have shown that patients with malignant melanoma with high NK cell activity display a reduced incidence of blood-borne metastasis. Conversely, reports from two centers have indicated a significantly greater risk of blood-borne metastasis in patients who smoke than in patients who do not. It is also noted that smokers have T lymphocyte profiles resembling those of patients with primary squamous carcinoma of the lung, and the extent of the change appears to be related to the extent of the disease. Thus, published work has linked cigarette smoking, immune system abnormalities, and prognosis in malignant melanoma. Smoking-induced immune system alterations are, however, usually reversible. The necessity of smoking cessation in postoperative malignant melanoma patients is emphasized.

84–1058. Chronic Diseases in Canada. **The Lung Cancer Epidemic Among Canadian Women.** *Chronic Diseases in Canada* 4(3): 33-35, December 1983.

During the period from 1932 to 1981, the age-standardized lung cancer mortality rate (age 25 to 74) for females in Canada increased from 2.8/100,000 to 25.1/100,000. Since the risk of developing lung cancer is strongly associated with smoking, the Canada Labour Force Survey smoking statistics were studied, and it was found that changes in smoking habits in the Canadian female population are similar to the increase in the female lung cancer rate. During the years from 1965 to 1979, the percentage of female regular smokers remained constant, but the proportion of heavy smokers continuously increased. In British Columbia, the percentage of heavy smokers grew more rapidly than in the other provinces, with a corresponding rise in lung cancer mortality in this province. The proportion of heavy smokers in British Columbia remains at a higher level than in other provinces, as does the rate of female lung cancer. Because of a conflict in these trends in other Canadian provinces, it is recommended that the pack-year stratified population attributable risk should be used as an alternative to the regular data collection method, which is based on observation of exposure and mortality trends.

84–1059. Dayal, H.; Kinman, J. **Epidemiology of Kidney Cancer.** *Seminars in Oncology* 10(4): 366-377, December 1983.

In a review of recent research, quantitative estimates of lifetime use of cigarettes and other tobacco products suggest a significant association between cigarette smoking and kidney cancer, but no correlation with consumption of other tobacco forms in The Third National Cancer Survey (7,518 cases). Large-scale prospective studies have found cigarette smoking to be a moderate risk factor for kidney cancer, particularly among heavy smokers. A number of case—control studies have observed a significant relationship between cigarette smoking and renal cell carcinoma (parenchymal, not including renal pelvis) in men, not only for cigarettes but also for other types of tobacco use, such as pipe or cigar smoking and tobacco chewing. Occupational studies have shown a significantly elevated risk for hypernephroma among coke plant workers, and several have suggested a synergistic effect between occupational exposure to cadmium and smoking. Both incidence and survival rates of kidney cancer have been increasing since the 1930s, but etiology of the disease remains poorly understood.

84–1060. Evans, D.A.P.; Eze, L. C.; Whibley, E. J. **The Association of the Slow Acetylator Phenotype With Bladder Cancer.** *Journal of Medical Genetics* 20(5): 330-333, October 1983.

Data indicate that there is an association between exposure to aromatic amines (those known to occur in tobacco smoke are polymorphically acetylated) and the development of bladder cancer. Data on 100 bladder cancer patients (only three were nonsmokers at time of diagnosis) were acetylator phenotyped, and statistically analyzed with four other series. Results show a significant association between the slow acetylator phenotype and bladder cancer (about 39 percent more with bladder cancer than the rapid acetylator phenotype). This association can be interpreted in two ways: (1) rapid acetylators may be protected against developing bladder cancer because they are better able to render amines noncarcinogenic by acetylation; or (2) slow acetylators have greater survival with bladder cancer than rapid acetylators. Further evidence will be required to differentiate between the alternatives. (Auth. Abs. Mod.)

84–1061. Gamble, J. F.; Ames, R. G. **The Role of the Lung in Stomach Carcinogenesis: A Revision of the Meyer Hypothesis.** *Medical Hypotheses* 11(3): 359-364, July 1983.

The theory that respiratory impairment serves as a gate to determine whether carcinogenic airborne particulates are retained in the lungs to produce lung cancer or cleared and swallowed to produce stomach cancer (the Meyer hypothesis) is reevaluated on the basis of an analysis of particle deposition patterns. Given the interactions between particle size, deposition location, and clearance, it is contended that airways obstruction is more likely to be related to increased clearance of the larger particles deposited in the tracheobronchial regions and to a decreased clearance of only the small particles (e.g., those found in cigarette smoke) deposited in the alveoli. Since dust of occupational origin tends to be of larger size, it is postulated that, regardless of the presence or absence of airways obstruction, a heavy smoker with high dust exposure is at increased risk of both lung and stomach cancer, while heavy smokers with low dust exposure are at risk of lung but not stomach cancer, and nonsmokers with high dust exposure are at risk only for stomach cancer.

84–1062. Gielen, J. E. **Toxicologie Genetique du Tabac. [Genetic Toxicology of Tobacco.]** *Revue Medicale de Liege* 38(17): 666-667, September 1, 1983, French.

The two main categories of chemical substances involved in carcinogenesis are (1) initiation agents responsible for the modification on the cellular gene level and (2) agents that promote accelerated transformation of a mutated cell into a malignant tumor. Cigarette smoke and tobacco condensates contain both chemical substances. The mutagenic and carcinogenic effects of cigarette smoke have been demonstrated in animal experiments, and there is some evidence to support similar effects in humans. Mutation products have been found in the urine of smokers, particularly aromatic amines derived from the naphthol nucleus. Complex DNA-benzopyrenes have recently been identified in the lungs of cancer patients who smoke. It is also noted that benzopyrene hydroxylase activity is found in the placentae of mothers who smoke. Finally, the number of circulating lymphocytes with chromosome aberrations is higher in smokers. It is concluded that components of cigarette smoke have indirect implications in human carcinogenesis.

84–1063. Greenberg, S. D. **Recent Advances in Pulmonary Cytopathology.** *Human Pathology* 14(10): 901-912, October 1983.

Benefits achieved by advances in pulmonary cytopathology over the past 5 years include detection and localization of occult lung cancers in high-risk cigarette smokers. Results of the Cooperative Early Lung Cancer Group, organized to screen a large cohort of high-risk male cigarette smokers (aged 45 years and over), demonstrated the value of techniques to detect, localize, and resect occult lung cancers when sputum specimens are positive for carcinoma but chest radiographs are normal. Occult lung cancers gradually evolve, over 20 to 30 years of cigarette smoking, from premalignant dysplasias to in situ and invasive carcinomas. From 80 to 90 percent of bronchial carcinoma cases in situ result from the generalized field effect of carcinogens inhaled in cigarette smoke, and a second (metachronous) primary tumor may eventually occur in 15 to 20 percent of cases. Etiology and suggested therapy are described. It is indicated that pulmonary cytologic findings in asbestos-associated lung cancers, which occur predominantly among asbestos workers who smoke, do not differ from those in nonasbestos-associated lung cancers. Additional advances in pulmonary cytopathology are described, as are related procedures, applications, and interpretation.

84–1064. Hellberg, D.; Valentin, J.; Nilsson, S. **Smoking as Risk Factor in Cervical Neoplasia. (Letter).** *Lancet* 2(8365/8366): 1497, December 24, 31, 1983.

Results of a case—control study (140 cervical neoplasias diagnosed during pregnancy, 280 controls) indicate an association between smoking and carcinoma in situ of the uterine cervix. Evaluation of the effect of smoking on preinvasive neoplasia (adjusted for known risk factors, male smoking, and socioeconomic status) showed highly significant associations ($p<0.001$) for female smoking and number of sexual partners, irrespective of other variables. Among cases, 113 of 140 women were either smokers or ex-smokers (80.7 percent); among controls, only 138 of 200 were smokers or ex-smokers (49.3 percent). When adjusted for male smoking, significance of female smoking remained unaffected. While further confirmation is essential, the case for a laboratory study of the relationship between smoking and cervical neoplasms is increasing. Two hypotheses are proposed: (1) a direct carcinogenic action of absorbed cigarette smoke; and (2) an immunosuppressant action of cigarette smoke.

84–1065. Higgins, I.T.T. **Commentary on "Possible Effects on Occupational Lung Cancer From Smoking Related Changes in the Mucus Content of the Lung".** *Journal of Chronic Diseases* 36(10): 677-680, 1983.

The hypothesis that smoking reduces the impact of respiratory carcinogens by increasing the mucus content of the lung (*Journal of Chronic Diseases* 36(10): 669-676, 1983) is disputed. Results of studies previously cited to show reduced lung dust burden in smokers compared with nonsmokers are described as less than convincing, and it is concluded that excess mucus in the lungs is a condition to be avoided by any possible means. The role of smoking as the most important cause of lung cancer and in the genesis of obstructive airways disease is emphasized; it is hoped that no one will be encouraged to smoke to reduce lung cancer risk from any occupational carcinogen. See also 84-1083.

84–1066. Hinds, M. W.; Kolonel, L. N.; Hankin, J. H.; Lee, J. **Dietary Vitamin A, Carotene, Vitamin C and Risk of Lung Cancer in Hawaii.** *American Journal of Epidemiology* 119(2): 227-237, February 1984.

Although smoking is known to be a major risk factor for lung cancer, it has been proposed that some dietary practices, such as the intake of vitamin A, carotene, and vitamin C may reduce the risk of such cancer. To test this hypothesis, a case—control study was conducted among the multiethnic population of Hawaii, subdivided into Japanese, Caucasian, Chinese, Filipino, and Hawaiian. Data were obtained by interviews with 364 primary lung cancer patients and 627 controls matched for age and sex (but not ethnic group). The most notable ethnic difference between cases and controls was among females, where 43.7 percent of cases but only 30.1 percent of controls were Caucasian, while the percentages among Japanese women were 21.3 and 38.2, respectively. These differences reflect the greater prevalence of cigarette smoking among Caucasian women compared with Japanese women in Hawaii. Separate analyses among all smokers ($n=653$) were conducted to determine the impact of dietary vitamin/carotene intake. An inverse relationship between total vitamin A/carotene intake and lung cancer risk was demonstrated for all males and for smoking males, but not for females. No evidence of an effect of dietary vitamin C on lung cancer risk was found in any group. Employment in a blue-collar occupation was also found to be a strong risk factor for lung cancer in these populations. (Auth. Abs. Mod.)

84–1067. Jozala, E.; Infante, S.; Marchini, J. S.; Okano, N. **Alcoolismo, Tabagismo e Carcinoma Epidermoide de Terco Medio do Esofago: Estudo Tipo Caso—Controle. [Alcohol, Tobacco and Middle Third Squamous Esophageal Cancer: A Case—Control Study.]** *Revista de Saude Publica* 17(3): 221-225, June 1983, Portuguese.

No association between squamous cell carcinoma of the middle third of the esophagus and tobacco use was demonstrated in a case—control study of outpatients at the Ribeirao Preto Medical School in Sao Paulo, Brazil. The 21 cases and 57 controls were matched by age, sex, residence, race, socioeconomic status, profession, and alcohol use as well as two smoking categories (from 1 to 20 cigarettes/day, or >20 cigarettes/day). Of the 19 cases for whom smoking histories were available, 16 were smokers, and most smoked <20 cigarettes/day; 42 controls smoked. The relative risk (RR) for smoking was 1.9 ($p<0.10$). A strong association (RR 26.7) was found for alcohol use.

84–1068. Kabat, G. C.; Wynder, E. L. **Lung Cancer in Nonsmokers.** *Cancer* 53(5): 1214-1221, 1984.

Among 2,668 patients with newly diagnosed lung cancer interviewed between 1971 and 1980, 134 cases occurred in "validated" nonsmokers. The proportion of nonsmokers among all cases was 1.9 percent (37 of 1,919) for men and 13.0 percent

(97 of 749) for women (sex ratio 1:2.6). Kreyberg Type II (mainly adenocarcinoma) was more common among nonsmokers, especially women, than among all lung cancer cases. Comparison of cases with an equal number of age-, sex-, race-, and hospital-matched nonsmoking controls showed no differences by religion, proportion of foreign-born, marital status, residence (urban/rural), alcohol consumption, or Quetelet index. Male patients had a higher educational level and included a higher proportion of professionals compared with controls. No differences in occupation or occupational exposure were seen in men. Among women, cases were 3.10 times as likely as controls to have worked in a textile-related job, but the significance of this finding is not clear. Preliminary data on exposure to passive inhalation of tobacco smoke, available for a subset of cases and controls, showed no differences except for more frequent exposure to sidestream tobacco smoke at work among male cases compared with controls. The need for more complete information on occupational and environmental exposure to tobacco smoke is discussed. (Auth. Abs. Mod.)

84–1069. Korsgaard, R.; Trell, E.; Simonsson, B.-G.; Svensson, G.; Linden, C.-J. **Genetisch Bedingte Lungenkrebsanfalligkeit bei Rauchern. [Genetically Controlled Susceptibility to Lung Cancer in Smokers.]** *Praxis und Klinik der Pneumologie* 37(1): 679-681, October 1983, German.

The possible relationship of aryl hydrocarbon hydroxylase (AHH) with smoking-associated respiratory tract cancers was studied in 112 cancer patients: 56 with small-cell bronchogenic cancer (all smokers), 32 with squamous cell carcinoma (all smokers), and 24 with adenocarcinoma (11 smokers). Induction of AHH in pulmonary lymphocytes by methylcholanthrene was measured fluorometrically and compared with noninduced lymphocytes. Significantly more patients with small-cell bronchogenic carcinoma and with squamous cell carcinoma showed high AHH inducibility (39 and 15, respectively) compared with the statistically expected number (4.3 and 2.4, respectively). However, patients with adenocarcinoma did not differ from expected levels of AHH inducibility. Data suggest that AHH is an important activator of aromatic hydrocarbons in respiratory tract carcinogenesis. While only small-cell bronchogenic carcinoma and pulmonary squamous cell carcinoma are clearly associated with smoking, AHH determination might be used to identify individuals at risk for developing lung cancer.

84–1070. Lawrence, C. E.; Paulson, A. S. **Cigarette Smoke: Cancer Risk at Low Doses.** In: Covello, V. T., Flamm, W. G.; Rodricks, J. V.; Tardiff, R. G. (Editors). *The Analysis of Actual Versus Perceived Risks.* Advances in Risk Analysis, Volume 1, New York, Plenum Press, 1983, pp. 169-174.

Nonsmokers experience an unwitting, involuntary exposure to cigarette smoke in the environment, and one method for establishing safe dose levels for such exposure is via extrapolation of data from high-dose experiments. A linear extrapolation technique was applied to data from a 1954 prospective epidemiologic study on deaths due to cancer of the lung and bronchus among 291,000 veterans. A high proportion of smokers in the sample had begun smoking before age 25, and observations of mortality were made for 8.5 years after administration of the questionnaire. To parallel animal experiments, the analysis was restricted to data on respondents who never quit smoking and smoked for a majority of their adult lives. The extrapolation yielded a virtually safe dose of 0.005 cigarettes/day, a level of environmental exposure that was exceeded in nonsmokers in nearly all cases. Thus, there is a large discrepancy between the levels to which involuntary exposure to other carcinogens are increasingly controlled and levels of passive exposure to cigarette smoke.

84–1071. Lilienfeld, A. M. **The Surgeon General's "Epidemiologic Criteria for Causality": A Criticism of Burch's Critique.** *Journal of Chronic Diseases* 36(12): 837-845, 1983.

The major areas of concern expressed in Burch's critique of the 1982 Surgeon General's Report (*Journal of Chronic Diseases* 36(12); 821-836, 1983) are reviewed and evaluated. In many instances, the original studies referred to are discussed. In re-examining the issue of the causal interpretation of the smoking—lung cancer relationship, the consistency, strength, coherence, and temporal nature of the association are emphasized. It is noted that smoking has never been considered the only cause of lung cancer, and that a one-to-one correspondence has not been claimed. However, given the body of data in its totality, there is a sufficiently high probability that the causal hypothesis is true, and that it is strong enough to warrant preventive action. See also 82-1299 and 84–1056.

84–1072. Lincoln, J. E.; Weinberg, G. B. **Re: "The Relationship Between the Geographic Distribution of Lung Cancer Incidence and Cigarette Smoking in Allegheny County, Pennsylvania." (Letters).** *American Journal of Epidemiology* 118(5): 783-784, November 1983.

An earlier theory that social class differences in smoking habits could account for differences in lung cancer mortality rates in Allegheny County, Pennsylvania (*American Journal of Epidemiology* 115(1): 40-58, January 1982), is questioned. The article reported current cigarette use in Allegheny County to be twice as high in lower occupational groups as in the higher ones, thus accounting for occupationally-related differences in lung cancer mortality. However, a 1953 national survey that included males and females under age 35 found differentials of only 20 percent. It is claimed that these social class differences in smoking were too small and had not been prevalent long enough. The original author notes that the smoking behavior documented represented the lifelong habits of four age groups, were local, and demonstrate dramatic differences in smoking by occupational categories. Given the excess mortality experienced in smokers compared with nonsmokers, differences in smoking prevalence of only 15 percent are not trivial. It is maintained that differential lung cancer mortality by geographic area or social class can be attributed to cigarettes. See also 82-0402.

84–1073. McLaughlin, J. K.; Mandel, J. S.; Blot, W. J.; Schuman, L. M.; Mehl, E. S.; Fraumeni, J. F., Jr. **A Population-Based Case—Control Study of Renal Cell Carcinoma.** *Journal of the National Cancer Institute* 72(2): 275-284, February 1984.

A population-based case—control study of renal cell carcinoma (495 cases and 697 controls) in the Minneapolis-St. Paul seven-county metropolitan area implicated cigarette smoking as a risk factor with an odds ratio (OR) among the 313 men of 1.6 (95 percent confidence intervals: 1.1 to 2.4) and among the 182 women of 1.9 (1.3 to 3.0). A statistically significant dose—response was observed in both sexes for pack-years of cigarette use. On the basis of calculations of attributable risk, it was estimated that 30 percent of renal cell cancers among men and 24 percent among women were due to smoking. High relative adult weight as measured by the body mass index (BMI) was found to be a major risk factor among women but not among men, with those in the highest 5 percent of the BMI having an OR of 5.9 (1.8 to 20.4) compared with the lowest quartile. Excess risks were also found for ethnic background (particularly German), long-term use of phenacetin-containing analgesics, heavy meat consumption, and tea consumption (females only) and for occupational exposure to petroleum, tar, and pitch products. Excesses of certain urologic and cardiovascular diseases were also observed among the cases compared with controls. (Auth. Abs. Mod.)

84–1074. Moller-Jensen, O.; Knudsen, J. B.; Sorensen, B. L.; Clemmesen, J. **Artificial Sweeteners and Absence of Bladder Cancer Risk in Copenhagen.** *International Journal of Cancer* 32(5): 577-582, November 15, 1983.

Tobacco smoking has been shown to be a significant risk factor for bladder cancer, and it has been proposed that a combination of smoking and other habits, such as use of artificial sweeteners, may increase the risk. A population-based case—control study of bladder cancer, including papillomas, was performed over a 2-year period in greater Copenhagen using 388 patients and 787 age- and sex-matched controls. The relative bladder cancer risk of 1.92 among male never smokers who used artificial sweeteners regularly was not significantly different from 1.0, with wide confidence limits from 0.47 to 7.84. The risk estimate became more stable when nonsmoking men and women were combined. Stratification for smoking differences between cases and controls did not change the conclusion of absence of risk associated with artificial sweeteners.

84–1075. Pepelko, W. E. **Experimental Respiratory Carcinogenesis in Small Laboratory Animals.** *Environmental Research* 33(1): 144-188, February 1984.

The experimental induction of respiratory tract tumors by inhalation of whole cigarette smoke in mice, rats, and hamsters is summarized. Although the epidemiological evidence linking smoking with lung cancer in humans is strong, success in inducing lung cancer in small laboratory animals was limited until the development of a sensitive strain of hamsters in 1979. The variations in responsiveness between humans and small animals and the characteristics of laboratory animals that moderate the effects of cigarette smoke are reviewed. Also discussed is lung cancer induction with organic chemicals (including benzo(*a*)pyrene), inorganic metallic compounds (including chromium), inorganic nonmetallic compounds, and other complex mixtures such as air pollution and engine exhaust fumes.

84–1076. Pollack, E. S.; Nomura, A.M.Y.; Heilbrun, L. K.; Stemmermann, G. N.; Green, S. B. **Prospective Study of Alcohol Consumption and Cancer.** *New England Journal of Medicine* 310(10): 617-621, March 8, 1984.

The apparent association between alcohol consumption and lung cancer found in a prospective study begun in the mid 1960s may be due to a residual confounding effect with cigarette smoking that could not be eliminated in the analysis. Of the Hawaiian Japanese men studied who drank more than 40 oz of alcohol/month, 66 percent were also current cigarette smokers compared with 41 percent in the remainder of the 7,837 subjects. The analysis, which was adjusted for the effects of age and cigarette smoking, revealed a positive association between consumption of alcohol and rectal cancer, accounted for primarily by an increased risk in men whose usual monthly consumption of beer was ≥ 500 oz (relative risk, 3.05; $p<0.01$ compared with those who did not drink beer). A significant positive relationship between alcohol consumption and lung cancer incidence was also found; subjects who consumed larger amounts of wine or whiskey had an increased risk compared with risks among nonconsumers of these beverages (relative risk, 2.19 ($p=0.03$) and 2.62 ($p<0.01$), respectively). No significant relationship between alcohol consumption and the incidence of stomach, colon, or prostate cancer was found. (Auth. Abs. Mod.)

84–1077. Radford, E. P. **Radiogenic Cancer in Underground Miners.** In: Boice, J. D., Jr.; Fraumeni, J. F., Jr. (Editors). *Radiation Carcinogenesis: Epidemiology and Biological Significance.* Progress in Cancer Research and Therapy, Volume 26, New York, Raven Press, 1984, pp. 225-230.

Data from multiple studies, which have yielded consistent results relating radon daughter exposure to lung cancer risk in underground miners, also show that the latent period before onset of cancer differs little between smokers and nonsmokers. However, in U.S. uranium miners there is a significant difference that may be the result of different rates of radon exposure. At high doses, as experienced by U.S. miners, there is a difference in time to excess cancer risk of 10 to 15 years for smokers compared with nonsmokers. At low doses, as experienced by Swedish iron miners, the difference is only 2 to 3 years. It is suggested that, at low doses, the accumulation of the initiation probability is the limiting factor in the onset of lung cancer. At high dose rates, the accumulation of initiation potential is so rapid that the time to onset may be determined by the presence of significant promoters; thus, cigarette smoking would have a strong influence on the time of development of cancer. The significance of the findings for radiogenic lung cancer, because of radon daughters detected in homes, is also noted.

84–1078. Repace, J. L. **Consistency of Research Data on Passive Smoking and Lung Cancer. (Letter).** *Lancet* 1(8375): 506, March 3, 1984.

A possible explanation is provided for the discrepancy in study results reported earlier (*Lancet* 2(8364): 1425-1426, December 1983) on the relationship between passive smoking and lung cancer risk in nonsmoking wives of smokers. It is suggested that the differences between the American study (179,739 women in the American Cancer Society cohort) and those conducted of Greek and Japanese wives may be related to the number of American women in the labor force (probably about 38 percent) who thus would have higher levels of smoke exposure compared with nonworking, nonsmoking wives. If the cohort is divided into working and nonworking controls and exposed subjects, the results may not be inconsistent with those of the other studies. Lung cancer deaths per 100,000 contributed by passive smoking are 2.25 for the home and 9.10 for the workplace. When such rates are applied to the subdivided groups, the overall risk ratio is similar to that found in the Japanese study. See also 84–0759.

84–1079. Rylander, R. **Environmental Tobacco Smoke. 3.7. Environmental Tobacco Smoke and Lung Cancer.** *European Journal of Respiratory Diseases* 65(Supplement 133): 127-133, 1984.

Although data suggest a relationship between environmental tobacco smoke (ETS) exposure and lung cancer, overall evaluation of available material from various studies does not establish ETS exposure-related increased risk for nonsmokers. While increased risk for lung cancer could be present under conditions with high exposure to ETS, it is concluded that the risk for an individual in the general population of nonsmokers would be very small. Methodological limitations are described for the studies discussed (Hirayama 1981; Trichopoulos et al. 1981 to 1983; Garfinkel 1981; Correa et al. 1983; Chan and Fung 1982; Gillis et al. 1983). It is suggested that future studies emphasize ETS dose description and preferably use the case—referent technique.

84–1080. Schachter, E. N. **Commentary on "Possible Effects on Occupational Lung Cancer From Smoking Related Changes in Mucus Content of the Lung".** *Journal of Chronic Diseases* 36(10): 681, 1983.

Evidence supporting the hypothesis that smoking, presumably through its bronchorrheic properties, may exert a protective effect (ascribed to related lack of penetration of respirable dusts) against occupationally induced lung cancer (*Journal of Chronic Diseases* 36(10): 669-676, 1983) is analyzed. While a dramatic incidence of lung cancer in chloromethyl ether-exposed workers is described for nonsmokers, a smaller incidence among smokers is still greater than would be expected in a cohort of smokers not exposed to chloromethyl ether. It is suggested that a high cancer incidence among ex-smokers should raise questions about the protective effect of smoking. With reference to additional studies cited, it is further indicated that (1) there is a relationship between absolute risk of respiratory cancer and a positive interaction between smoking and arsenic exposure; and (2) there is clearcut evidence for a relationship between lung cancer and positive interactions between smoking and asbestos/uranium exposure. See also 84-1083.

84–1081. Schmidt, F.; Trichopoulos, D. **Passive Smoking and Lung Cancer. (Letters).** *Lancet* 1(8378): 684, March 24, 1984.

Rebuttal is made to critical comments (*Lancet* 2(8362): 1309, December 3, 1983) regarding reports linking passive smoking and lung cancer. While the methodology in one study is imperfect, the evidence that nonsmoking wives with lung cancer were far more likely to live with smokers (61.5 percent, nearly three times the expected figure) and had no other apparent attributable risk factor is incontrovertible. In the other study, the crude relative risk was incorrectly given, but the statistics and conclusions are correct. See also 84-0757.

84–1082. Shirname, L. P.; Menon, M. M.; Nair, J.; Bhide, S. V. **Correlation of Mutagenicity and Tumorigenicity of Betel Quid and Its Ingredients.** *Nutrition and Cancer* 5(2): 87-91, 1983.

Tumorigenicity studies in Swiss mice treated with aqueous extracts of betel quid (BQ), betel quid with tobacco (BQT), betel nut (BN), and betel leaf (BL) showed that BN and BQ induced lung tumors (47 and 26 percent, respectively). However, when BN was fed with BL, tumorigenicity was lowered to 38 percent. BL alone was not tumorigenic. Thus, the mutagenicity of betel quid and its ingredients is correlated with tumorigenicity. The mutagenic activity of betel quid and its ingredients was also determined using *Salmonella typhimurium* test strains TA 100, TA 1535, TA 98, and TA 1538, in both the presence and the absence of S9 mixture. BQ, BQT, and BN were mutagenic in strain TA 100. BL was not mutagenic in any of the four strains. Arecoline and arecaidine, which are major alkaloids present in BN, were mutagenic in all four test strains. (Auth. Abs. Mod.)

84–1083. Sterling, T. D. **Possible Effects on Occupational Lung Cancer From Smoking Related Changes in the Mucus Content of the Lung.** *Journal of Chronic Diseases* 36(10): 669-676, 1983.

Increased mucus in the lungs of smokers may help in elimination of dust and toxic substances by facilitating migration of particles and aerosols, and may add protection by thickening the mucus layer. Magnetometric measurements indicate that lungs of chronically exposed miners and millers contain less dust if they smoke than if they do not. Epidemiological studies of lung cancer among occupations that expose workers to dusts or toxic aerosols have reported a larger prevalence of lung cancer among nonsmoking than among smoking workers. A recent study using beagle dogs reported significantly fewer respiratory tumors among animals exposed to radon, radon daughters, uranium dust, and tobacco smoke than among animals similarly exposed except for tobacco smoke. The hypothesis that increasing mucus in the lungs of exposed workers may protect against lung disease needs verification in appropriate animal studies. If protective qualities of lung mucus are verified, means that will increase mucus flow in nonsmoking (and smoking) workers exposed to respiratory hazards need to be explored. (Auth. Abs. Mod.)

84–1084. Trell, E.; Trell, L.; Janzon, L.; Laurell, P.; Korsgaard, R. **Verbesserung der Motivation zur Rauchewohnung bei Individuen mit Genetischen Risikofaktoren fur Lungenkrebsentwicklung. [Improved Motivation for Giving Up Smoking in Persons With Genetically Determined Risk of Cancer of the Respiratory Tract.]** *Praxis und Klinik der Pneumologie* 37(1): 687-690, October 1983, German.

The level of the inducible intracellular enzyme system, aryl hydrocarbon hydroxylase (AHH) was determined in lymphoblast cultures from middle-aged smokers and nonsmokers in a preventive medical program in Malmo, Sweden. AHH induction, which is genetically determined and appears to be of importance for the development of smoking-related cancers of the airways and mouth, was not influenced by smoking habits. The high AHH inducers among the smokers (8.6 percent of the smokers studied) were invited to a group-mediated antismoking information and counseling program. Preliminary results regarding feasibility and success were promising. (Auth. Abs.)

84–1085. Winn, D. M.; Ziegler, R. G.; Pickle, L. W.; Gridley, G.; Blot, W. J.; Hoover, R. N. **Diet in the Etiology of Oral and Pharyngeal Cancer Among Women From the Southern United States.** *Cancer Research* 44(3): 1216-1222, March 1984.

Cigarette smoking, snuff-dipping, and alcohol consumption were strongly associated with oral and pharyngeal cancer incidence in a case—control interview study of North Carolina women. The possible protective effects of a diet high in fruits and vegetables were investigated among 227 female oral/pharyngeal cancer patients and 405 matched female controls classified by age, race, education, tobacco and alcohol use, wearing of dentures, and use of mouthwash. The relative risk (RR) for cigarette smoking only was 1.5; the RR for alcohol and cigarettes was 3.2; and snuff and cigarette use combined had an RR of 3.7. The combined risk for all three habits was 2.8. The relationship between snuff-dipping and cigarette smoking was evident among low, moderate, and high consumers of each food group studied (fruits/vegetables, breads/cereals, and meat/fish). High, but not moderate, intake of fruits and vegetables was associated with a reduced RR in nonsmokers, while decreasing RRs were found with increasing intake of fruits and vegetables in those who used cigarettes only or cigarettes and snuff. The overall reduction in risk with greater fruit and vegetable consumption that was observed across all groups was consistent with the hypothesis that vitamin C and/or β-carotene intake is associated with a reduced risk of oral and pharyngeal cancer.

84–1086. Wynder, E. L. **Some Thoughts on Preventive Oncology.** *Progress in Clinical and Biological Research* 132E: 339-345, 1983.

Up to 80 percent of human cancers may be attributed to environmental factors; specifically, tobacco, alcohol, diet, and occupational and other exposures. Cigarette smoking alone is related to one-third of male cancer deaths, while among women the figure is 8 percent and rising. Cigarettes are associated principally with neoplasms of the lung, vocal cords, bladder, and pancreas; cigar and pipe smoke influence the risk of oral, laryngeal, and esophageal cancer. Tobacco chewing also increases the risk of cancer of the oral cavity. Prevention of cancer will require motivating the individual to adopt a healthy lifestyle, which involves a strategy of health education from early childhood. Product modification, especially in the yields of cigarettes, and reduced dietary fat and increased fiber intake would also be helpful, although to what extent these changes would effect cancer incidence is unknown. Legislation to increase the drinking age, improve the content of processed foods, and establish maximum permissible tar and nicotine yields of cigarettes is recommended. Obstacles to prevention include human apathy (crisis always seems to motivate more people than prevention) and marketing by industries that profit from such goods as tobacco, alcohol, and high-fat foods and who advertise their wares aggressively.

84–1087. Wynder, E. L. **Preventive Oncology: An Opportunity for Clinical Cancer Centers. (Letter).** *Journal of the National Cancer Institute* 72(2): 201-202, February 1984.

Although lifestyle factors such as tobacco use are unquestionably linked to cancer, broad societal trends toward more healthful behavior patterns have not occurred. Only among white males has there been a reduction in smoking. Reasons given for the persistence of such habits include the lack of enthusiasm for behavior change and little involvement in health promotion by the medical community and other obstacles to health outside the medical profession (such as the marketing of cigarettes). The oncological community is urged to find strategies for providing preventive services within the confines of clinical cancer centers. It is noted that smoking cessation programs require minimal investment, and could eventually become economically self-sufficient.

NON—NEOPLASTIC RESPIRATORY DISEASES

84–1088. Andrion, A.; Pira, E.; Mollo, F. **Pleural Plaques at Autopsy, Smoking Habits, and Asbestos Exposure.** *European Journal of Respiratory Diseases* 65(2): 125-130, February 1984.

Data on postmortem findings of pleural plaques (PP), smoking habits, and occupational history of asbestos exposure showed a significant association between PP and smoking among middle-aged and elderly men. Correlations were studied for 898 adults (478 smokers) from the general population. No significant excess of PP was found among the 50 ex-smokers. Among the 269 women (54 smokers), 20 evidenced PP. Although pleural changes were found more frequently in smokers than in nonsmokers, no significant association was observed. When occupational history was considered, the association was present in both exposed and nonexposed men over 50 years of age. No association occurred in nonexposed women, and in those exposed, the absolute number of cases was too small to evaluate any relationship. Consumption, in pack-years, did not correlate significantly with the frequency of PP. Autopsy data confirmed the association between occurrence of PP and smoking habits. (Auth. Abs. Mod.)

84–1089. Bake, B. **Environmental Tobacco Smoke. 3.1. Does Environmental Tobacco Smoke Affect Lung Function?** *European Journal of Respiratory Diseases* 65(Supplement 133): 85-87, 1984.

While data strongly suggest an association between exposure to environmental tobacco smoke (ETS) in the home and a small reduction in ventilatory lung function in adults exposed more than 15 to 20 years, the potential magnitude of ETS exposure is uncertain since no study has considered the combined effects of various related elements. It is indicated that, if all other factors are equal, exposure to this agent would probably also affect lung function in children; however, a review of various studies reflects uncertainty regarding parental smoking effects and only minute consequences.

84–1090. Barlow, P. B.; Nelson, E. C.; Howland, J.; Meier, F. A.; Brooks, A. H.; Brent, P. L.; Parker, H. W.; Secker-Walker, R. H. **Locating Community Residents With Chronic Airway Obstruction. A Comparison of Four Strategies.** *American Review of Respiratory Disease* 129(3): 361-365, March 1984.

In the course of locating patients with chronic airway obstruction (COAD) to participate in an educational intervention program, implementation of four different strategies yielded differing information on smoking status. Patients located by an advertising campaign smoked least (35 percent) at the time of baseline assessment and had the highest percentage of never smokers (33 percent). Patients located by hospital records showed the highest proportion of smokers who had quit (44 percent). Over half (51 percent) of patients located by mailed questionnaire smoked at the time of baseline assessment. The strategies did not appear biased with regard to the sex of patients located. The excess of males among patients located by hospital records is consistent with the higher prevalence of COAD among men, while the high rate of cigarette smoking among patients located by physician referral and questionnaires is probably due to a selection bias. The higher rate of smoking cessation among patients located by hospital records suggests that as patients grow older and/or more impaired by airway obstruction, they are more likely to quit smoking. In patients selected by the advertising campaign, the relatively large number of never smokers may indicate a high level of concern for personal health.

84–1091. Berend, N. **Small Airways Disease.** *Australian and New Zealand Journal of Medicine* 13(4): 393-397, August 1983.

Airway inflammation, the most important lesion found in small airways disease, may be the precursor to the subsequent development of emphysema and is an important cause of functional abnormalities in smokers. A current hypothesis suggests that small airways disease may be detected early in the smoking careers of subjects who later develop chronic respiratory disability. In these persons, complete cessation of smoking should be the primary therapeutic goal. With the recent development of special tests to assess small airways function, disease can be detected in asymptomatic smokers who may have normal routine lung function tests. Most of the tests to assess airway function, however, are also influenced by other physiological factors, such as loss of elastic recoil. The hypothesis that early small airways disease leads to chronic disability cannot be confirmed until results of long-term epidemiological studies are available. Without such confirmatory evidence, routine use of these special tests cannot be advocated. (Auth. Abs. Mod.)

84–1092. Bjartveit, K.; Foss, O. P.; Gjervig, T. **The Cardiovascular Disease Study in Norwegian Counties. 5. Smoking, Cough and Phlegm.** *Acta Medica Scandinavica* Supplement 675: 61-81, 1983.

Data on smoking habits, smoking-related symptoms and diseases, and variations in prevalence in three Norwegian counties are presented, gathered in the first screening of a cardiovascular disease study started in 1974, based on 57,859 adults aged 20 to 49. In males, the percentage of daily smokers differed only slightly between age groups, with the number of ex-smokers increasing with age. In females, the number of daily smokers decreases with increasing age, the proportion of ex-smokers remaining fairly constant. Consumption is higher in males than in females, and higher for both sexes in Finnmark (the northern county) than in the two southern counties: 66.6

percent of males aged 45 to 49, and 46.4 percent of females are daily smokers in Finnmark, compared with 51.0 and 23.8 percent in Sogn og Fjordane, and 53.4 and 35.2 percent in Oppland, respectively. Smoking rates are higher in men reporting coronary heart disease compared with the healthy group; however, for myocardial infarction cases, the proportion of smokers is lower, reflecting a high number of ex-smokers. Mean serum cholesterol concentration and serum triglycerides are highest in smokers. In women, mean systolic and diastolic blood pressure and mean relative weight are highest for nonsmokers; in men, the same tendency is found for diastolic, but not for systolic blood pressure. Cough and phlegm appear with higher frequency in men than in women and, when adjusted for level of consumption, geographic differences disappear. Cough and phlegm are also more frequent in areas of high air pollution, and higher in men than in women, suggesting that men are more heavily exposed to polluting agents at work. Pulmonary/pleural/hilar chest X-ray changes increase regularly with increasing cigarette consumption, as does the percentage of persons on sick leave. In males, the number is 2.5 times higher in those smoking ≥ 20 cigarettes/day. Results confirm that smoking is by far the most important environmental factor determining the level of cough and phlegm frequency in Norway. A key to prevention is clearly demonstrated after cessation of smoking, when symptom frequency soon returns to the level of nonsmokers. An overview of the complete study is presented in 84-1139.

84–1093. Brown, P. J.; Greville, H. W.; Finucane, K. E. **Asthma and Irreversible Airflow Obstruction.** *Thorax* 39(2): 131-136, February 1984.

Duration and severity of asthma and smoking history were studied in 42 male and 47 female chronic asthmatics (38 current or ex-smokers) with irreversible airflow obstruction. All patients were treated with theophylline and a beta agonist for 4 weeks; a group of 48 patients showing little initial improvement was also treated with prednisolone for the final 2 weeks. Forced expiratory volume in 1 sec (FEV_1) was measured at 0, 2, and 4 weeks. No significant differences between patients who were current or ex-smokers and those who had never smoked were observed. The mean difference between the predicted normal FEV_1 and the highest value observed during treatment was 0.29 l, ($p<0.001$). Similar findings were noted when results for nonsmokers and those whose asthma started in adult life were analyzed separately, suggesting that development of irreversible obstruction in asthma is not dependent on smoking or on a childhood onset of asthma.

84–1094. Buczko, G. B.; Day, A.; Vanderdoelen, J. L.; Boucher, R.; Zamel, N. **Effects of Cigarette Smoking and Short-Term Smoking Cessation on Airway Responsiveness to Inhaled Methacholine.** *American Review of Respiratory Disease* 129(1): 12-14, January 1984.

Data indicate that cigarette smoking is associated with increased airways responsiveness to inhaled methacholine in a dose-related manner. Threshold of airway responsiveness to methacholine aerosol was determined in 53 apparently healthy persons. In 18 nonallergic nonsmokers matched according to sex and age to 18 nonallergic smokers, the mean methacholine threshold of airway response (T), as measured using partial flow-volume curves, had a tendency to be greater in nonsmokers, but the difference was not significant for the group as a whole. However, it was significant for a subset of nine matched pairs with a cigarette consumption greater than 10 pack-years (mean T nonsmokers, 2.8 mg/ml; smokers, 0.3; $p=0.036$). In 17 smokers who stopped smoking for an average of 99 days, T was not significantly different, although the majority reported improvement of a variety of respiratory symptoms after cessation. (Auth. Abs. Mod.)

84–1095. Buczko, G. B.; Zamel, N. **Combined Effect of Cigarette Smoking and Allergic Rhinitis on Airway Responsiveness to Inhaled Methacholine.** *American Review of Respiratory Disease* 129(1): 15-16, January 1984.

Among matched pairs who underwent methacholine challenge, smokers with allergic rhinitis had a lower threshold airway response compared with similar smokers without allergic rhinitis. Nonatopic subjects were compared with atopic subjects with allergic rhinitis who were otherwise healthy. All were matched for sex, age, and smoking habits; 15 pairs were nonsmokers and 11 pairs were smokers. Threshold of airway response to inhaled methacholine, based on partial flow-volume curves, was not significantly different between nonatopic nonsmokers and nonsmokers with allergic rhinitis. However, smokers with allergic rhinitis had a threshold on the average of 3 doubling doses lower than smokers without allergic rhinitis. It is concluded that chronic cigarette smoking and allergic rhinitis have a combined effect on nonspecific airway responsiveness. (Auth. Abs. Mod.)

84–1096. Chan-Yeung, M.; Buncio, A. D. **Leukocyte Count, Smoking, and Lung Function.** *American Journal of Medicine* 76(1): 31-37, January 1984.

Leukocyte counts were significantly higher among all current smokers (1,550) compared with ex-smokers (1,161) and nonsmokers (735) in two different work populations in British Columbia. The interrelationships between leukocyte counts, cigarette smoking, and pulmonary function were examined in 1,826 white male workers in a pulp and paper mill and 1,620 white male workers in an aluminum smelter. Workers took part in epidemiologic health studies that consisted of a medical and occupational questionnaire, spirometric measurements, and leukocyte counts. Measurements of air contaminants in the work environment were also carried out simultaneously by personal and area sampling. No significant increase in the prevalence of respiratory symptoms and lung function abnormalities was found between the exposed and nonexposed workers in each work population. Similarly, no difference in leukocyte count was found between exposed and nonexposed workers. In both populations, there was an inverse correlation between leukocyte count and forced expiratory volume in 1 sec and forced vital capacity of workers regardless of smoking habits, suggesting that the leukocyte response to external stimuli may be another determinant of lung function measurements. (Auth. Abs. Mod.)

84–1097. Clark, T.J.H. **Airways Obstruction. Treatment of Airways Obstruction.** *Postgraduate Medical Journal* 59(Supplement 3): 54-61, 1983.

Chronic obstructive bronchitis is a major cause of airways obstruction, and to date, cigarette smoke inhalation is the most important factor in pathogenesis. Treatment for airways obstruction must be guided by understanding of pathophysiology and pathogenesis of the underlying diseases. Inhalation of cigarette smoke causes acute airway narrowing, but chronic airways obstruction occurs only in a minority of susceptible persons in whom the rate of decline in measures of lung function is greater than normal. The pathogenesis of emphysema is similar to that of chronic obstructive bronchitis, except for possible involvement of a genetic deficiency in alpha$_1$-antitrypsin. The first approach to these disorders is prevention, based on health education and encouragement of smokers to quit. Once the condition is present, treatment may involve use of bronchodilators, drug therapy, diuretic therapy (with a diagnosis of cor pulmonale), or surgery. Specific treatment approaches that may be employed in cases of chronic obstructive bronchitis, emphysema, and asthma are reviewed.

84–1098. Cohen, N.; Measham, A. R.; Akbar, J. **Smoking and Respiratory Disease Symptoms in Rural Bangladesh.** *Public Health* 97(6): 338-346, November 1983.

In the two rural villages studied, respiratory disease symptoms in males at all age levels showed a relationship with smoking, with most smokers reporting "cough." Most heavy smokers and all women smokers smoked bidis; 71 percent of men but only 1 percent of women were smokers. Smoking expenditures accounted for up to 5 percent of household income. One of the lowest socioeconomic groups had the highest consumption levels. It is noted that there must be other causal factors for respiratory symptoms in women; morning phlegm and wheezing were two to three times more frequent among women who never smoked compared with male nonsmokers. Smoke from cooking fires and respiratory infections in childhood are suggested as possible factors. Respiratory disease will increase in prevalence if smoking incidence increases, although improving immunization coverage and the treatment of respiratory infections in childhood would be useful preventive strategies. The most urgent public health measure needed is to reduce the prevalence of smoking.

84–1099. Edwards, C.; Carlile, A.; Rooke, G. **The Larger Bronchi in Byssinosis: A Morphometric Study.** *Journal of Clinical Pathology* 37(1): 20-22, January 1984.

While smoking among workers exposed to cotton dust had deleterious effects on the course of byssinosis. no impact on bronchial morphology was found. The proportions of gland, muscle, and cartilage were measured in the named bronchi of 43 workers with byssionosis and compared with proportions in normal subjects ($n=5$) and chronic bronchitics ($n=18$). Smoking habits and symptoms of byssinotics were also investigated. In byssinotics, proportions of gland, muscle, and cartilage were higher than in normal, nonsmoking subjects at all levels in the bronchial tree, and were similar to those in chronic bronchitics. The increase was not confined to the lobar bronchi. Although no morphologic differences were noted, smokers experienced first symptoms 8.5 years earlier, left work 7.0 years earlier, and died 10 years earlier than nonsmoking contemporaries ($p<0.025$, 0.0125, and 0.0005, respectively). It is concluded that although exposure to cotton dust is a factor in the pathogenesis of chronic byssinosis, anatomical changes in the bronchi of Lancashire cotton workers are nonspecific and are confounded by other environmental factors. (Auth. Abs. Mod.)

84–1100. Edwards, J. H.; AlZubaidy, T. S.; Altikriti, R.; Bunny, H. **Byssinosis. Inhalation Challenge With Polyphenol.** *Chest* 85(2): 215-217, February 1984.

Contrary to previous reports, inhalation challenge with a condensed polyphenol isolated from cotton failed to induce symptoms such as those experienced by employees returning to work at the cotton mill after a weekend break. A total of 29 male workers were tested; 11 smoked >10 cigarettes/day, 7 were ex-smokers, and 11 were nonsmokers. Lung function test results did not differentiate between polyphenol and saline challenges. However, significant differences in pulmonary function were observed with respect to byssinosis and smoking habits. The nonsmoking normal controls had significantly greater lung function values than nonsmoking byssinotic subjects. Differences between the smoking controls and smoking byssinotics approached but did not reach significance. Ex-smokers showed patterns similar to nonsmokers.

84–1101. Ellis, E. F. **Asthma in Childhood.** *Journal of Allergy and Clinical Immunology* 72(5, Part 2): 526-539, November 1983.

Risk factors for asthma in children include parental smoking, a history of atopy, acute lower respiratory tract disease, and bronchiolitis or croup. Smoking by parents has been associated with lower forced expiratory flow rates in children. Most studies report a preponderance of asthma in boys over girls, with ratios varying from 1.3:1 to 3.3:1. Signs of the disease may be apparent in the first 2 years of life and are often associated with viral respiratory infections. Disproportionate narrowing of peripheral airways and decreased static elastic recoil properties of the lung predispose infants and young children to asthma. During midchildhood there is a tendency toward improvement, with continued improvement during adolescence. The goal of management of the child with asthma is to reduce symptoms sufficiently so that the child can regularly attend school, engage in play activities, and sleep through the night uninterrupted, while avoiding unacceptable levels of adverse drug effects. Nonpharmacologic management includes both avoidance of environmental irritants and behavioral approaches to overcome emotional precipitants that lead to attacks. Pharmacologic treatment includes the use of four classes of drugs: (1) adrenergics, drugs: (2) theophylline, drugs: (3) cromolyn, and (4) corticosteroids. The prognosis of asthma in childhood is good. However, a history of such respiratory problems in childhood substantially increases the risk of developing chronic obstructive airways disease in adults who smoke. (Auth. Abs. Mod.)

84–1102. Finkelstein, M. M.; Vingilis, J. J. **Radiographic Abnormalities Among Asbestos-Cement Workers. An Exposure-Response Study.** *American Review of Respiratory Disease* 129(1): 17-22, January 1984.

Smokers were at higher risk for both pleural and parenchymal radiographic lung abnormalities in a longitudinal study of 181 asbestos-cement workers. Of subjects with a known smoking status, 74 percent were current smokers. The relationships between the 32-year risks of developing abnormalities, smoking habits, and cumulative exposure to asbestos dust were analyzed with log-normal curves. Smokers had higher risks of both abnormalities than did nonsmokers (relative risk approximately 3). In this cohort, men leaving asbestos exposure had a risk of radiographic progression similar to men continuing in exposure, and men with abnormal radiographs had higher mortality rates than did men whose radiographs were normal. (Auth. Abs. Mod.)

84–1103. Galdston, M.; Levytska, V.; Schwartz, M. S.; Magnusson, B. **Ceruloplasmin. Increased Serum Concentration and Impaired Antioxidant Activity in Cigarette Smokers, and Ability to Prevent Suppression of Elastase Inhibitory Capacity of Alpha$_1$-Proteinase Inhibitor.** *American Review of Respiratory Disease* 129(2): 258-263, February 1984.

Data indicate that (1) cigarette smoking can cause partial inactivation of serum antioxidant activity accompanied by insufficient compensatory increase in ceruloplasmin concentration, and (2) ceruloplasmin may protect the lung against oxidant(s) in cigarette smoke and air pollutants. Serum was analyzed to determine (1) ceruloplasmin concentration and antioxidant activity (percentage inhibition of autoxidation of ox-brain homogenate) in healthy male and female smokers and nonsmokers; and (2) the capacity of ceruloplasmin to prevent suppression of the elastase inhibitory capacity of α_1-proteinase inhibitor by the oxidant chloramine T and by cigarette smoke solution. Mean ceruloplasmin concentration was 18 percent higher in 35 female smokers than in 46 male smokers ($p<0.001$); 17 percent higher in 22 female nonsmokers than in 18 male nonsmokers ($p<0.005$); 15 percent higher in the female smokers than in the female nonsmokers ($0.02<p<0.01$); and 14 percent higher in the male smokers than in the male nonsmokers ($p<0.001$). Serum antioxidant activity showed significant linear correlations with serum ceruloplasmin in smokers and nonsmokers of both sexes; correlation coefficients, all significant, ranged from 0.65 to 0.50. For comparable ceruloplasmin concentrations, serum antioxidant activity was significantly lower in smokers (males: 9 percent, $p<0.001$; females: 7 percent, $0.05<p<0.01$) than in nonsmokers. There was a linear relationship between ceruloplasmin concentration and its ability to prevent suppression of the elastase inhibitory capacity of α_1-proteinase inhibitor by chloramine T and cigarette smoke solution. (Auth. Abs. Mod.)

84–1104. Gibson, J. B.; Martin, N. G.; Oakeshott, J. G.; Rowell, D. M. **Lung Function in an Australian Population: Contributions of Polygenic Factors and the *Pi* Locus to Individual Differences in Lung Function in a Sample of Twins.** *Annals of Human Biology* 10(6): 547-556, November-December 1983.

In an investigation of various causes of impaired lung function in 203 Australian twin pairs aged 18 to 34 years, an effect of smoking was detected, but accounted for less than 3 percent of the variance, even after adjusting for age and height. Twins were classified as smokers if they currently smoked cigarettes or had given up smoking only since their last birthday. Consumption was rated as cigarettes smoked/day (from 0 to 40), and three smoking variables were considered: years smoked, consumption rating, and the product of the two variables. Measures of lung function detected significant genetic variation in females and males, but no evidence of family environmental effect. However, systematic environmental differences affecting lung function were noted between males, so that heritability was lower in males than in females. A measure of deviation in lung function from that found in healthy nonsmokers (forced expiratory volume (FEV)) tended to decrease with increasing cigarette consumption. The relative risk of females having an FEV value in the bottom 5 percent of the sample was more than doubled in heavy smokers compared with nonsmokers.

84–1105. Gouveia, J.; Hercend, T.; Gros, F.; Dorval, T.; Hazon, J.; Misset, J. L.; Schwarzenberg, L.; Ribaud, P.; Lemaigre, G.; Santelli, G.; Reizenstein, P.; Gaget, H. **Correlation Between Precancerous Bronchial Metaplasia and Cigarette Consumption, and Preliminary Results of Retinoid Treatment.** *Cancer Detection and Prevention* 5(4): 461-466, 1982.

Despite the subjectivity of estimates of consumption among 106 heavy smokers, smoking was found to be significantly ($p<0.02$) and positively correlated to the index of bronchial metaplasia. Subjects (15 females, 91 males) ranged in age from 29 to 71 (mean 49.8) and had at least 15 pack-years of smoking. Healthy blood donors ($n=60$) served as controls. Of the 101 evaluated by bronchoscopic examination and multiple biopsies, 52 had sufficiently pronounced metaplasia to require treatment, and received 25 mg ET$_1$ (an aromatic retinoid) daily for 6 months. Vitamin A and its derivatives (retinoids) can prevent squamous metaplasia induced not only by vitamin A deficiency but also by carcinogenic hydrocarbons. An aromatic retinoid such as ET_1 has been shown to prevent chemically induced papillomas in mice and to amplify certain immunologic reactions. The index of metaplasia was significantly ($p<0.01$) reduced in 30 patients who underwent repeat bronchoscopy following the 6-month treatment. It is noted that 85 percent of the women had a low index of metaplasia compared with 42 percent of the men, although there was no significant difference in reported cigarette consumption. (Auth. Abs. Mod.)

84–1106. Graham, B. L.; Dosman, J. A.; Cotton, D. J.; Weisstock, S. R.; Lappi, V. G.; Froh, F. **Pulmonary Function and Respiratory Symptoms in Potash Workers.** *Journal of Occupational Medicine* 26(3): 209-214, March 1984.

While data show that potash workers ($n=1,663$) had significantly better forced expiratory volume in 1 sec/forced vital capacity (FEV_1/FVC) and maximal mid-expiratory flow rate values compared with community controls, nonsmokers ($n=478$) had significantly higher values than smokers ($n=799$). No significant interaction between smoking and working in the potash industry was found for pulmonary variables tested. Among current smokers, however, there was a small but statistically significant decline in percentage of expected FEV_1/FVC associated with number of years employed. Compared with a nonexposed control group, potash workers had higher prevalences of cough, dyspnea, and chronic bronchitis but better pulmonary function. Prevalence of symptoms and pulmonary function abnormalities were similar among workers at the four mines tested and at the various job locations. Potash dust, diesel fumes, and other air contaminants may have an irritant effect that leads to the increased prevalence of cough and chronic bronchitis. Although no adverse effects of the potash mine environment on pulmonary function were found, results reflect a healthy worker effect or some selection process that makes the potash workers appear healthier in a cross-sectional study. (Auth. Abs. Mod.)

84–1107. Greaves, I. A.; Wegman, D. H.; Smith, T. J.; Spiegelman, D. L. **Respiratory Effects of Two Types of Solder Flux Used in the Electronics Industry.** *Journal of Occupational Medicine* 26(2): 81-85, February 1984.

The lack of association between cigarette smoking and respiratory symptoms reported by electronic workers who soldered printed circuit boards was probably due to a "healthy worker" effect, resulting from individuals with lower than average lung function who left the industry early in the course of employment. Questionnaires and lung spirometry were administered to 104 electronics workers (93 women, 11 men), of whom 68 had worked only with a rosin-core solder, and 36 who had changed from rosin- to an aqua-core solder 15 months before the study. For combined groups, 35 percent were current smokers, 10 percent ex-smokers, and 55 percent nonsmokers. Symptoms of eye, throat, and nose irritation occurred in nearly half of the total group. Lower respiratory tract symptoms, including cough, phlegm production, and wheezing, also occurred with increased frequency, compared with reported rates among a general population. Similar symptom rates were observed for both groups. It is concluded that symptoms were work-related, since subjects improved on weekends or during vacation periods. On average, lung spirometry performed before starting work on Monday mornings showed no impairment in lung function; over the course of the workday there were small decrements in the forced expiratory volume at 1 sec and forced vital capacity, but these decrements were unrelated to the individual amounts of solder used.

84–1108. Herrmann, H. **Die Bedeutung Kommunaler Umweltfaktoren fur die Entstehung Chronischer Atemwegserkrankungen. [The Importance of Environmental Factors on the Development of Chronic Lung Diseases.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(2): 163-176, May 1983, German.

A longitudinal study of lung function performed on men in two East German towns, one (Halle) highly industrialized and polluted and the other (Schwerin) much less polluted, indicated that smoking was a more significant factor in the development of bronchitis and chronic bronchitis than air pollution. In 1971, clinical symptoms of bronchitis were found in Halle men in 14.3 percent of nonsmokers, 13.4 percent of ex-smokers, and 17 percent of smokers. Among Schwerin men, the percentages were 6.5, 7.8, and 11. In 1980, Halle figures were 17.9, 16.0, and 22.6; for Schwerin men, 16.9, 13.4, and 14.2. In 1971, chronic bronchitis was diagnosed in Halle men in 15.1 percent of nonsmokers, 19.2 percent of ex-smokers, and 25.6 percent of smokers. In 1980, Halle percentages were 14.2, 15.5, and 22.0; Schwerin figures were 3.9, 9.8, and 17.1. Light smokers (<24 g tobacco/day) registered the same incidence of chronic bronchitis in industrial and nonindustrial areas, while among consumers of 25 to 50 g/day, chronic bronchitis incidence was higher in rural than in industrial areas. Also, the annual decrease in lung function was less rapid among ex-smokers in the rural area compared with the industrial area.

84–1109. Holt, P. G.; Turner, K. J. **Environmental Tobacco Smoke. 3.5. Respiratory Symptoms in the Children of Smokers: An Overview.** *European Journal of Respiratory Diseases* 65(Supplement 133): 109-120, 1984.

A review of the association between parental smoking habits and respiratory symptoms in offspring concludes that infants whose parents smoke experience a greater frequency of respiratory infections, particularly in the first year of life. Also, increased wheeze, exacerbation of asthma, and reduced lung function in older children have been reported. These effects appear to be more strongly related to maternal than to paternal smoking, especially when infants are involved. As a tool to assist in future research, a scheme of interrelated immunological and inflammatory processes that could serve as a mechanism of induction of respiratory symptoms in children exposed to environmental tobacco smoke (ETS) is proposed. It is recommended that new research focus on (1) the actual absorption of ETS by children; (2) the effects of lower doses, in the range relevant to children; and (3) the age-dependent variation in tobacco smoke sensitivity, using younger laboratory animals.

84–1110. Hughes, J. A.; MacArthur, A. M.; Hutchison, D.C.S.; Hugh-Jones, P. **Long Term Changes in Lung Function After Surgical Treatment of Bullous Emphysema in Smokers and Ex-Smokers.** *Thorax* 39(2): 140-142, February 1984.

The annual rate of change in lung function was estimated in a group of 11 patients (6 ex-smokers and 5 smokers) who received surgical treatment for bullous emphysema and were followed by regular assessment for a minimum of 4 years. For each patient, the annual rate of change in forced expiratory volume in 1 sec (FEV_1) and "relaxed" vital capacity were estimated before and after bronchodilator aerosol and in carbon monoxide transfer factor (TLCO), transfer coefficient (KCO), and arterial carbon dioxide and oxygen tensions ($PaCO_2$ and PaC_2). While all lung function variables declined at a faster rate in patients who continued to smoke than in ex-smokers, the difference in rate was significant for FEV_1 (before bronchodilator), TLCO, and KCO. In ex-smokers, no significant change in most lung function indices was observed. In smokers, all lung function indices except $PaCO_2$ declined at a rate significantly greater than zero. Findings suggest that long-term results of surgical treatment for bullous emphysema are probably greatly improved if patients stop smoking. (Auth. Abs. Mod.)

84–1111. Hurst, T. S.; Graham, B. L.; Cotton, D. J. **Fast vs. Slow Exhalation Before O_2 Inhalation Alters Subsequent Phase III Slope.** *Journal of Applied Physiology* 56(1): 52-56, January 1984.

Data show that rapid exhalation to residual volume (RV) in the sitting position increased the phase III slope in smokers, an effect opposite to that seen in nonsmokers, possibly due to peripheral airways obstruction. A group of 10 symptom-free lifetime nonsmokers and 17 smokers, all with normal pulmonary function, performed single-breath N_2 washout tests by either exhaling slowly (slow maneuver) from end inspiration (EI) to RV or exhaling maximally (fast maneuver) from EI to RV. After either maneuver, subjects slowly inhaled 100 percent O_2 to total lung capacity (TLC) and without breath-holding, exhaled slowly back to RV. In nonsmokers seated upright, phase III slope of single-breath N_2 test ($\Delta N_2/1$) was lower for the fast maneuver compared with the slow, but this difference disappeared when maneuvers were repeated in the supine position. In contrast, $\Delta N_2/1$ was higher for the fast maneuver compared with the slow in smokers seated upright. For the slow maneuver, $\Delta N_2/1$ was similar for smokers and nonsmokers, but for the fast maneuvers, $\Delta N_2/1$ was higher in smokers than in nonsmokers. It is suggested that the fast exhalation to RV decreases $\Delta N_2/1$ in normal subjects by decreasing apex-to-base differences in regional ratio of RV to TLC (RV/TLC), but increases $\Delta N_2/1$ in smokers because regional RV/TLC increases distal to sites of small airways obstruction when the expiratory flow rate is increased. (Auth. Abs. Mod.)

84–1112. Huuskonen, M. S.; Husman, K.; Jarvisalo, J.; Korhonen, O.; Kotimaa, M.; Kuusela, T.; Nordman, H.; Zitting, A.; Mantyjarvi, R. **Extrinsic Allergic Alveolitis in the Tobacco Industry.** *British Journal of Industrial Medicine* 41(1): 77-83, February 1984.

The relationship between occupational exposure to raw tobacco dust and clinical, radiological, functional, and immunological evidence of extrinsic allergic alveolitis was examined in 57 Finnish tobacco factory workers. The working environment of all workers indicated exposure to spores of different molds, while 29 workers (51 percent) showed antibodies against one or more of the microbes. Work-related respiratory symptoms were observed in 15 (26 percent) of the workers, while 8 (14 percent) showed slight radiographic pulmonary fibrosis. Spirometry showed a tendency toward restriction and obstruction, especially in small airways. Diffusion capacity was decreased in 18 percent of the workers. Three clinical cases of typical allergic alveolitis were also found. Findings suggest that exposure to spores of various molds (especially *Aspergillus fumigatus*) in the manufacture of tobacco products may induce symptoms and signs relating to extrinsic allergic alveolitis. (Auth. Abs. Mod.)

84–1113. Jackson, S.H.D.; Cruickshank, J. K.; Beevers, D. G.; Bannan, L. T. **Ethnic Differences in Peak Expiratory Flow Rate in Birmingham Factory Workers.** *Postgraduate Medical Journal* 59(696): 671-673, October 1983.

Data on 698 Birmingham factory workers showed ethnic differences in mean height, peak expiratory flow rates (PEFR), and smoking habits for the 95 Asians, 255 blacks, and 348 whites studied. Only 21.3 percent of the Asian males and 5.0 percent of the Asian females were smokers, compared with 53.9 and 15.2 percent for blacks and 50.4 and 49.3 for whites, respectively. Asian men tended to have lower PEFR than black or white men when age, height, and smoking habits were accounted for. This trend reached statistical significance in Asian females, despite the small sample size ($n=20$). There were no significant differences in PEFR between blacks and whites of either sex. It is concluded that there is a genuine effect of ethnic group in the results among Asian women.

84–1114. Janoff, A.; Niewoehner, D. E.; Davies, S. F. **Urine Desmosine Excretion. (Letters).** *American Review of Respiratory Disease* 129(3): 511-512, March 1984.

A previous analysis of urine desmosine excretion and lung function (*American Review of Respiratory Disease* 128(3): 473-475, September 1983) is criticized for using subjects that were occupationally exposed to inhaled pollutants (firefighters). Chronic occupational exposure could have produced elevated desmosine excretion rates that masked increases associated with smoking and smoking-related lung damage. However, if lung elastolysis occurs at a constant low rate over many years, immunologic measurement of elastin degradation products may not be sensitive enough to detect lung damage in smokers. In reply, it is stated that since ventilatory function in the firefighters was above population norms, use of this group did not constitute a serious confounding factor. Thus, the higher levels of urine desmosine obtained do not appear to be related to occupation, and the authors remain skeptical as to the potential value of desmosine in diagnosing emphysema. See also 84-0133.

84–1115. Jedrychowski, W. **Biological Meaning of the Prospective Epidemiological Study on Chronic Obstructive Lung Disease and Aging.** *Archives of Gerontology and Geriatrics* 2(3): 237-248, November 1983.

Comparison of respiratory symptoms after a 5-year period among a representative sample of Cracow inhabitants (1,096 men and 1,476 women, 19 to 70 years old) showed that the overall relative risk of chronic obstructive lung syndrome related to smoking was 7.3 for men and 1.8 for women; logistic curves were slightly different in men and women when smoking was considered. The steepest increase with age was observed among male smokers, and the contrast between smokers and nonsmokers tended to be more pronounced in males over age 50. Both male and female smokers had a faster forced expiratory volume in 1 sec decline with age than nonsmokers. Excluding the confounding effects of smoking and respiratory symptoms, ventilatory lung function deteriorated twice as rapidly in subjects over age 50, compared with younger groups. It is suggested that particles with a diameter smaller than 1 μm affect gradual functional deterioration of lung function in smokers and the sensory receptors in the air pathway. Chronic effects of smoking include increased respiratory tract secretion and a narrowing of the air passage. While data may suggest that males are more susceptible than females, the average male smoker sampled began smoking at age 19 and averaged 20 cigarettes/day, compared with onset at age 22 and 15 cigarettes/day for women. Contrary to expectations, data show that the elderly run a substantial risk of developing chronic obstructive lung disease even when no respiratory symptoms are present and they are nonsmokers. It is concluded that changes produced by relatively mild pulmonary symptoms, such as productive cough, added to preexisting aging-related changes may lead to obstructive lung disease.

84–1116. Kandt, D. **Zur Reproduzierbarkeit von Engebnissen Bronchialer Reaktivitatsprufungen. [Reproducibility of Results of the Bronchial Reactivity Tests.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 162(1): 30-35, 1984, German.

Lung function tests were administered to 383 outpatients (average age 48 years), with various degrees of bronchial hyperactivity. Vital capacity and forced expiratory volume in 1 sec (FEV_1) were measured before and 5 and 10 minutes after inhalation of a nebulized histamine solution. Measurements were reported over a 3- to 6-year period, and other factors were considered. Bronchial hyperactivity returned to normal (as tested by inhalation provocation) in 48.5 percent of 97 nonsmokers, but only in 31.1 percent of the 193 patients who smoked, a statistically significant difference. Clearly, smokers maintain bronchial hyperreactivity longer than nonsmokers. Clinical symptoms of hyperreactivity and age were also significant factors.

84–1117. Kimmel, E. C.; Diamond, L. **The Role of Nicotine in the Pathogenesis of Pulmonary Emphysema.** *American Review of Respiratory Disease* 129(1): 112-117, January 1984.

A single, relatively high dose of nicotine did not induce or augment the development of pulmonary emphysema in Long-Evans rats. In an investigation of the potential role of nicotine in the pathogenesis of pulmonary emphysema, the acute lethality and the pulmonary toxicity of a single, endotracheally administered dose of nicotine were studied. The 24-hour LD_{50} of nicotine was established at 19.3±5.4 mg/kg. Subsequently, rats were treated with either nicotine alone (3 or 7.5 mg/kg), porcine pancreatic elastase (PPE) alone (20 IU/kg), or PPE (200 IU/kg) followed 5 days later by nicotine (3 mg/kg). The selected dose of PPE was below the threshold dose for induction of emphysematous lesions. Cage and sham-treated control groups were also included. Four weeks after treatment, extensive physiologic tests were conducted to evaluate the ventilatory, mechanical, and gas exchange functions of the lungs, and lung tissue specimens were taken for estimation of tissue volume density. Although a few isolated incidents of significant intergroup differences were observed, no distinctive pattern of functional or structural change reminiscent of emphysema was noted in any of the experimental groups. (Auth. Abs. Mod.)

84–1118. Kueppers, F. **Proteasen und Proteaseinhibitoren; Atiologische Rolle bei Chronischobstruktivem Emphysem. [Proteases and Protease Inhibitors: Their Role in Chronic Obstructive Pulmonary Disease.]** *Praxis und Klinik der Pneumologie* 37(1): 700-702, October 1983, German.

While smoking, particularly cigarettes, plays a major role in emphysema etiology, with equal exposure to smoke, the resulting lung damage may differ greatly. In addition, aggregation of emphysema in families has been known for a long time. The "Protease" or "Elastase Hypothesis" attempts to combine these factors by noting that proteolytic enzymes are the agents responsible for the destruction of lung tissue in the development of emphysema. Normally, proteases and their inhibitors are in equilibrium. A disturbance of this equilibrium in favor of proteases leads to destruction of lung tissue, emphysema, and airways obstruction. Tobacco smoke can alter the equilibrium in two ways: (1) oxidizing substances in smoke can inactivate alpha-1-antitrypsin; and (2) smoking frequently maintains a chronic bronchitis that increases the number of leukocytes, and hence granulocytic elastase, in the lung. The same conditions are met in the inherited disease of alpha-1-antitrypsin deficiency, which results in a 90 percent decrease in protease inhibitor. (Auth. Abs. Mod.)

84–1119. Labedzki, L.; Magnussen, H.; Hartmann, V.; Loos, U.; Mackes, G.; Schwabe, H.; Braunschweig, D.; Tippelmann, G.; Frost-Hensel, E. **Bronchuslavage bei Lungenkrankheiten: Zytologie und Makrophagenfunktion. [Bronchoalveolar Lavage in Persons With Lung Disease: Cytology and Macrophage Function.]** *Praxis und Klinik der Pneumologie* 37(1): 832-835, October 1983, German.

Bronchoalveolar lavage revealed cell counts of 15.2 x 10^6 in nonsmokers, 21.3 x 10^6 in smokers, 18.4 x 10^6 in bronchogenic carcinoma patients, and 32.4 x 10^6 in sarcoidosis patients. In the latter subjects, 28.4±6.6 percent lymphocytes were counted. There was no significant difference between the groups in adherence and phagocytosis. Killing of *Candida guillermondii* was significantly reduced in smokers compared with nonsmokers and persons with bronchogenic carcinoma or sarcoidosis. Chemotaxis in smokers was significantly increased compared with nonsmokers. Chemotaxis in sarcoidosis patients was increased, with migration up to 2.8 percent in some cases, which could be of diagnostic importance if verified by further studies. (Auth. Abs. Mod.)

84–1120. Lebowitz, M. D. **Environmental Tobacco Smoke. 3.3. The Effects of Environmental Tobacco Smoke Exposure and Gas Stoves on Daily Peak Flow Rates in Asthmatic and Non-Asthmatic Families.** *European Journal of Respiratory Diseases* 65(Supplement 133): 90-97, 1984.

Environmental tobacco smoke (ETS) showed no effect on daily peak flows (Vmax) or symptoms in children or adults, asthmatics, or others in a study of 117 families (229 subjects) in Tucson. Monitoring was conducted over a 2-year period, using daily diaries for symptom information and peak flows, measured daily with a flow meter. Indoor carbon monoxide was significantly associated with gas stove usage, but not with ETS, gas furnaces, or other gas appliances. Children exposed to ETS had a nonsignificantly higher average Vmax, but Vmax was negatively affected in adult smokers and ex-smokers. Indoor total suspended particulates (TSP) had an effect on daily symptoms in adults and on Vmax in asthmatics that must be associated with characteristics of indoor TSP not correlated with ETS.

84–1121. Lebowitz, M. D.; Knudson, R. J.; Burrows, B. **Family Aggregation of Pulmonary Function Measurements.** *American Review of Respiratory Disease* 129(1): 8-11, January 1984.

Data from 271 nuclear families of the Tucson epidemiologic study of airway obstructive diseases (AOD) indicated that parental passive smoking had no effect on children's pulmonary function measurements. Of the parental pairs and natural children, all of whom had satisfactory pulmonary function data, 72.3 percent of fathers, 50 percent of mothers, and 1.7 percent of children over 15 years were ever smokers. Parameters measured were forced vital capabity (FVC), forced expiratory volume, maximal expiratory flows after 50 and 75 percent of the FVC had been expired, ponderal index, history of obstructive airway disease, and family size. No significant interaction between smoking habits and spousal lung function was found. There was significant product—moment correlation of pack-years between fathers and their children and fathers with their spouses, and a borderline correlation of mother—son smoking. No correlation of smoking with body size or habitat was noted. Initial regression analysis showed significant correlations of the pulmonary function variables after controlling for age and sex. Body habitus, as measured by the ponderal index, was highly aggregated as well. Pulmonary function measurements were aggregated in families independent of family size, reported diagnosed AOD, and children's smoking, even though both asthma and smoking showed significant familial aggregation. After controlling for the familial aggregation of body habitus, a major determinant of pulmonary function, there was no remaining independent aggregation of pulmonary function measurements. (Auth. Abs. Mod.)

84–1122. Levine, R. J.; DalCorso, R. D.; Blunden, P. B.; Battigelli, M. C. **The Effects of Occupational Exposure to the Respiratory Health of West Virginia Morticians.** *Journal of Occupational Medicine* 26(2): 91-98, February 1984.

The presence of pulmonary function abnormalities and chronic bronchitis among 90 morticians was greatest among current smokers (n=32), intermediate in ex-smokers (n=30), and lowest among never smokers (n=28). Standardized respiratory disease questionnaires and pulmonary function tests were administered to licensed morticians attending an educational program. Detailed occupational histories were also obtained, including estimates of the number of bodies personally embalmed. Pulmonary function of morticians compared favorably with residential populations in Oregon and Michigan. Relatively high occupational exposure was not associated with chronic bronchitis or pulmonary function deficits. Comparison of smoking-specific, age-matched exposure groups also revealed no correlation between high exposure and the prevalence of cough, phlegm, or wheeze. Results suggest that long-term intermittent exposure to low levels of formaldehyde gas exerts no significant effect on respiratory health. (Auth. Abs. Mod.)

84–1123. Lindberg, E.; Hedenstierna, G. **Chrome Plating: Symptoms, Findings in the Upper Airways, and Effects on Lung Function.** *Archives of Environmental Health* 38(6): 367-374, November-December 1983.

No synergistic effect was found between smoking and exposure to chrome plating among 100 workers (64 smokers, 40 nonsmokers) exposed to chrome plating and 119 (67 smokers, 52 nonsmokers) controls, for whom respiratory symptoms, lung function, and changes in the nasal septum were noted. Complaints of nasal irritation were common among subjects whose average daily exposure exceeded 1 $\mu g/m^3$ of chromic acid, while the frequency of chronic bronchitis was similar to that in different control series. Nasal septal ulceration and perforation were seen in two-thirds of the subjects exposed to peak levels of 20 $\mu g/m^3$ or more. Both forced vital capacity and forced expiratory volume in 1 sec were reduced by 0.2 l. The forced mid-expiratory flow diminished by 0.4 l/sec from Monday morning to Thursday afternoon in subjects exposed to a daily average of 2 $\mu g/m^3$ or more chromic acid, but there was no clear correlation with short-term high exposure. This Monday—Thursday reduction in lung function was less apparent in smokers compared with nonsmokers, possibly due to varying smoking habits and selection effects. (Auth. Abs. Mod.)

84–1124. Lundberg, J. M.; Martling, C.-R.; Saria, A.; Folkers, K.; Rosell, S. **Cigarette Smoke-Induced Airway Oedema Due to Activation of Capsaicin-Sensitive Vagal Afferents and Substance P Release.** *Neuroscience* 10(4): 1361-1368, 1983.

Exposure of rats to smoke from one cigarette caused local edema due to a marked increase in vascular permeability from the epiglottis down to bronchioli, as indicated by extravasation of Evans blue in the airway mucosa. The cigarette smoke-induced extravasation was still present after removal of the tar and nicotine content of the smoke, suggesting that chemical irritants in the vapor phase were the main mediators of the vascular permeability response. Local or systemic pretreatment with capsaicin or (D-Arg^1, D-Pro^2, D-Trp^{7,9}, Leu^{11}) SP, a substance P antagonist, abolished or significantly reduced the airway edema induced by cigarette smoke or vagal nerve stimulation. No reduction of the smoke- or vagally-induced tracheal edema was seen upon pretreatment with mepyramine plus cimetidine, fentanyl, disodiumchromoglycate, methylprednisolone, or terbutaline. Results indicate that the smoke- or vagally-induced tracheal edema is most likely to be due to substance P release from local capsaicin-sensitive afferent neurons in the airway mucosa. Local administration of substance P antagonists may be considered as a pharmacological means of inhibiting local mucosal edema in the airways caused by airway irritants such as cigarette smoke. (Auth. Abs.)

84–1125. Marcelle, R. **Les Bronchopneumopathies. [The Bronchopneumopathologies.]** *Revue Medicale de Liege* 38(17): 663-665, September 1, 1983, French.

The four categories of respiratory system effects caused by various components of tobacco smoke are (1) nonspecific cellular changes, (2) carcinogenesis in the epithelium, (3) hyperexcitability of smooth muscles in the bronchi, and (4) reinforcement of hypoxemia linked to preexisting cardiorespiratory insufficiency. Tobacco smoke has been shown to modify the structure of certain cells in the respiratory system, which can lead to chronic bronchial disease and emphysema. Most people affected by this frequent and sometimes severe disease (7 percent of deaths) are heavy smokers. Cessation of smoking can reverse the damage in the precancerous stage of epithelial cancer development, and while only about 2/1,000 smokers die of bronchopulmonary cancer, 98 percent of such cancers are found in smokers. The hyperexcitability of smooth muscle fibers in the bronchial system caused by tobacco smoke produces bronchial asthma, or symptoms resembling asthma that may be related to the histamines released by the smoke. Because the carbon monoxide in tobacco smoke has an affinity for hemoglobins, the blood of some smokers contains up to 15 percent carboxyhemoglobin, leading to loss of oxygenation. Such hypoxemia may be tolerated by a healthy person, but people with reduced cardiorespiratory function may suffer angina or other symptoms. Tobacco use should be discouraged in people with chronic heart and lung diseases.

84–1126. Murphy, R.L.H., Jr.; Gaensler, E. A.; Holford, S. K.; Del Bono, E. A.; Epler, G. **Crackles in the Early Detection of Asbestosis.** *American Review of Respiratory Disease* 129(3): 375-379, March 1984.

In an evaluation of the usefulness of chest auscultation as a method for screening industrial populations exposed to asbestos, it was found that weighted crackle score (WCS) in a group of 386 workers was associated with pack-years of cigarette smoking. The interaction of smoking history, years of exposure, and crackle index was statistically evaluated using logistic regression and both a trichotomous and dichotomous response variable. Using the trichotomous response variable, age appeared to be the most significant factor; the smoking—asbestos interaction was nonsignificant. The smoking effect was present only when comparing a WCS of 1 or 2 with a WCS of 0, suggesting that the WCS of 1 or 2 may be the result of smoking, while a WCS of 3 or higher is associated with asbestosis. Using the dichotomous model and comparing a WCS of 0, 1, or 2 with a WCS of 3 or higher, an asbestos effect was seen, but no smoking effect was found. In comparing a WCS of 0 versus >1, a smoking effect was observed, but no asbestos effect was found and the age effect was again highly significant. Results of the dichotomous model confirmed those of the trichotomous model.

84–1127. Nakamura, M.; Hildebrandt, J. **Cigarette Smoke Acutely Increases Collateral Resistance in Excised Dog Lobes.** *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 56(1): 166-174, 1984.

The acute effects of cigarette smoke or drug inhalation on collateral conductance (Gcoll) were studied in freshly excised dog lobes held at fixed volumes. A double-lumen catheter was wedged into a segmental bronchus, and air, smoke, or aerosol flowed into the blocked segment at a constant pressure of 2 cmH_2O. A capsule glued over a small area of perforated pleura of the segment was used to measure alveolar pressure; the capsule could also be used to measure small airway flow (Vcap) through the segment. Gcoll was almost linearly dependent on lung volume, rising about fivefold between 20 and 100 percent inflation (30 cmH_2O). During smoke inhalation, Gcoll began decreasing almost immediately, roughly halving with the first cigarette and falling to about 20 percent after two cigarettes. Similar proportions were obtained at other lung volumes. Pulmonary conductance (oscillator) in the remainder of the lobe decreased only modestly to 78 percent of control after two cigarettes. In lobes exposed to 4.5 percent CO_2 after air, Gcoll rose 25 to 50 percent, but Vcap increased only 5 to 10 percent. However, acetylcholine chloride aerosol reduced both flows by similar ratios. Isoproterenol did not prevent or reverse smoke-induced collateral constriction but did reverse the effects of acetylcholine on both pathways. Results suggest that in excised lungs, aerosols acted on larger segmental airways in series with collateral channels and with peripheral airways, whereas CO_2, and particularly cigarette smoke, provoked more marked effects on the most distal smooth muscle. (Auth. Abs.)

84–1128. Novo, S.; Davi, G.; Caimi, G.; Castello, F.; Valdes, L.; Marino, S.; Romano, A.; Fazio, M.; La Menza, B.; Mazzola, A.; Carpentiere, G.; Sarno, A.; Strano, A. **Platelet Activity in Relation to Smoke and Exercise in Patients With Chronic Obstructive Lung Disease: Effects of Platelet Antiaggregating Drugs.** *Advances in Experimental Medicine and Biology. Thrombosis and Cardiovascular Diseases* 164: 339-357, 1980.

Data from 10 chronic obstructive lung disease patients (average age 57.1 years) indicate that both physical exercise and cigarette smoking activate platelet function, demonstrated by greater betathromboglobulin release and higher thromboxane B_2 levels. Such activation is antagonized by dipyridamole infusion and by aspirin pretreatment followed by dipyridamole infusion. Prostacyclin (PGI_2) release is increased by exercise and inhibited by cigarette smoke. At high doses, aspirin completely inhibits PGI_2 synthesis; dipyridamole has no effect. Exercise tends to increase pulmonary ventilation, demonstrated by a reduction in plasma carbon dioxide (CO_2); smoking tends to diminish pulmonary ventilation, increase plasma CO_2, and reduce pH; dipyridamole increases the effect of exercise and inhibits the effect of smoking. Similar results occur after aspirin pretreatment, and therefore may depend on a direct spasmolytic dipyridamole action, independent of prostaglandins. Aspirin may also reduce the action of some intermediate endoperoxides that stimulate the smooth muscle contraction of the respiratory tract and increase airways resistance. (Auth. Abs. Mod.)

84–1129. Peters, J. M.; Smith, T. J.; Bernstein, L.; Wright, W. E.; Hammond, S. K. **Pulmonary Effects of Exposures in Silicon Carbide Manufacturing.** *British Journal of Industrial Medicine* 41(1): 109-115, February 1984.

Radiographic findings indicate that lung opacity appears about 20 years earlier in ever smokers than in never smokers. Information on smoking status, present cigarette consumption for smokers, and years since cessation for ex-smokers was obtained for 200 workers at a silicon carbide manufacturing plant. Smoking categories were defined as nonsmoker, ex-smoker, and current low (<15 cigarettes/day), medium (15-to-25 cigarettes/day), and high (>25 cigarettes/day) consumption. Lifetime exposure to respirable particulates is closely related to frequency of opacities for both smokers and nonsmokers, with smokers generally showing a higher frequency of opacity than nonsmokers. Average lifetime exposure to respirable particulates was strongly associated with profusion category, especially for round opacities. Cumulative exposure to respirable particulates strongly affected both forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV_1) in ever smokers, while cumulative sulfur dioxide exposure was also related to FEV_1 (but not to FVC) in ever smokers. Duration of employment was significantly related to both FVC and FEV_1 in ever smokers, but not in never smokers. It is concluded that smoking enhances the pulmonary effect of exposure to respirable particulates and sulfur dioxide. Findings did not yield reliable information on the proposed synergistic interaction between silicon dioxide and particulates.

84–1130. Phelan, P. D. **Does Adult Chronic Obstructive Lung Disease Really Begin in Childhood?** *British Journal of Diseases of the Chest* 78(1): 1-9, January 1984.

The most important aspect of childhood and its relationship with adult lung disease is the acquisition of the addiction to cigarette smoke. Smoking is the major etiological factor in chronic hypersecretory bronchitis, although it does not develop in all smokers. A smaller percentage of adults, almost all of whom are smokers, develop progressive obstructive lung disease, frequently associated with hypersecretory bronchitis. While cessation of smoking may reduce the rate of deterioration in lung function, lost function is not recovered. A review of other studies does not support the hypothesis that lower respiratory infection in childhood is a risk factor for adult chronic obstructive lung disease. Recurrent "bronchitis" in infancy, which is almost certainly a manifestation of asthma, frequently continues into adult life, and thus may be associated with continuation of respiratory symptoms. It is possible that bronchial hyperreactivity may be associated with more rapid progression of chronic obstructive disease, but this suggestion requires confirmation by long-term studies. Prevention of smoking, however, is more urgent than further attempts to define the relationship between childhood and adult lung disease.

84–1131. Poloni, M.; Mento, S. A.; Mascherpa, C.; Ceroni, M. **Value of Spirometric Investigations in Amyotrophic Lateral Sclerosis.** *Italian Journal of Neurological Sciences* 4(1): 39-46, April 1983.

Pulmonary function in patients with amyotrophic lateral sclerosis (ALS) was investigated as a function of several parameters, including smoking status, to obtain clinical data on respiratory muscle impairment. The sample included 37 male ALS patients classified as smokers, ex-smokers, or nonsmokers;

15 of these subjects received serial spirometric tests for varying periods (5 until death). Analysis of smoking status showed a nonsignificant increase in residual volume in heavy smokers or ex-smokers. Vital capacity, total lung capacity, Motley Index, and forced expiratory volume in 1 sec were more impaired in nonsmokers. The increase in residual volume noted in smokers and ex-smokers was probably related to an obstructive syndrome and is also common in chronic inflammatory bronchopulmonary forms. It is concluded that neither smoking nor pulmonary/bronchial pathology in the present or past history significantly influence spirometric indexes in ALS.

84–1132. Rebohle, E. **Berufliche Schadstoffexposition und Deren Bedeutung fur die Entstehung Obstruktiver Chronischer Lungenkrankheiten. [Exposure to Occupational Harmful Substances and Its Significance for the Development of Chronic Obstructive Lung Disease.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(3): 266-272, 1983, German.

Results of East German epidemiological studies indicate that smoking and occupational exposures are leading risk factors for chronic obstructive bronchitis and chronic respiratory diseases, and display additive effects. Higher incidence of chronic bronchitis in middle-aged male workers compared with female workers from 1976 to 1978 is attributed to greater predominance of smoking among males. Since the smoking factor cannot be differentiated from the occupational influence, recommended approaches to prevention of occupational obstructive lung diseases include (1) not exceeding the maximum allowed concentrations of occupational noxious agents, and (2) antismoking information programs.

84–1133. Schindl, R. **Einfluss der Umweltfaktoren auf die Lungenfunktion Screeninguntersuchung mit Flow-Volumen bei Schulkindern in Stadt und Land. [Influence of Environmental Factors on Pulmonary Function. Screening Investigations With Flow-Volume Among School Children in Town and Countryside.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(2): 199-203, May 1983, German.

The interaction between environmental factors (including passive smoking) and long-term air pollution was determined by measuring pulmonary function in 3,482 Austrian schoolchildren in an urban area with high sulfur dioxide air concentrations, a suburban housing development, and a rural area. Passive smokers comprised 21, 10.5, and 13 percent, respectively, of the students. Maximum expiratory flow at 25 percent values among smokers and passive smokers were higher in the rural and suburban populations than among urban students. Cough, dyspnea, sulfur dioxide emission, and dust were also correlated with passive smoking.

84–1134. Teculescu, D. B.; Bohadana, A. B.; Peslin, R.; Cereceda, J. V. **1-Second Forced Expiratory Volume and Density Dependence in Early Airflow Limitation.** *Respiration* 44(6): 433-438, November-December 1983.

Results indicate that density dependence (when using simple measures) is not as sensitive as originally anticipated for detecting airflow limitation in its early phase. Density dependence variables (helium-to-air difference in forced expiratory flows at 50 and 25 percent vital capacity (VC), and volume of isoflow) were compared with spirographic performance (VC, forced expiratory volume in 1 sec (FEV$_1$)) in men considered at risk and in those with early airflow limitation. Subjects were 76 men aged 33 to 56 years; 30 asymptomatic nonsmokers, 22 asymptomatic smokers with normal spirographic performance, 17 smokers with chronic bronchitis, and 7 chronic bronchitis patients (1 nonsmoker) with minimal obstructive ventilatory impairment. Low level correlations (coefficients of <0.3) occurred between ΔHe percent or volume of isoflow and FEV$_1$ or FEV$_1$/VC. Possible confounding factors for the correlations are discussed, and it is concluded that density dependence variables are not consistently related with FEV$_1$ in subjects at risk or with minimal airflow limitation. It is noted that a previous study was unable to find any relationship between amount of tobacco used (either current consumption or total pack years) and density dependence in asymptomatic smokers. (Auth. Abs. Mod.)

84–1135. Thiemann, H.-H. **Luftverunreinigung und Pravalenz Unspezifischer Erkrankungen der Atemorgane im Kindesalter. [Air Pollution and Prevalence of Nonspecific Bronchopulmonary Diseases in Childhood.]** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(2): 209-217, May 1983, German.

Although the prevalence of nonspecific bronchopulmonary diseases in children from various regions of East Germany paralleled the level of air pollution, 3.72 percent of children of smokers had chronic or recurrent bronchitis, compared with 3.45 percent of nonsmokers' children. Children in various German regions were studied. In addition to pollution levels, factors influencing the prevalence of children's chronic nonspecific lung disease were air temperature, humidity, sulfur dioxide level, and dust.

84–1136. Trell, E.; Janzon, L.; Lindell, S.-E.; Korsgaard, R.; Simonsson, B. G.; Svensson, G.; Linden, C.-J. **Rauchgewohnheiten, Kohlenmonoxydhamoglobin und Lungenkrankheiten. [Smoking Habits, Carboxyhemoglobin and Airways Disease.]** *Praxis and Klinik der Pneumologie* 37(1): 673-675, October 1983, German.

As part of a preventive medical program, the smoking habits, carboxyhemoglobin levels, and symptoms of chronic obstructive pulmonary disease (including knowledge of the association with smoking and "quitting smoking" motivation) were assessed in middle-aged men in Malmo, Sweden. The COHb levels and pulmonary symptoms were clearly correlated with smoking habits. Whereas a general knowledge of the dangers associated with smoking did not influence the desire or success of reducing or stopping smoking in those who still smoked at the time of the medical screening program, the presence and recognition of smoking-related airways symptoms did. This observation suggests the inclusion of measurements of carbon monoxide in the expired air in regular nurse-mediated antismoking counseling, in addition to preventive medical screening in the campaign in Malmo.

84–1137. Ware, J. H.; Dockery, D. W.; Spiro, A., III.; Speizer, F. E.; Ferris, B. G., Jr. **Passive Smoking, Gas Cooking, and Respiratory Health of Children Living in Six Cities.** *American Review of Respiratory Disease* 129(3): 366-374, March 1984.

Maternal cigarette smoking was associated with increases of 20 to 35 percent in the rates of eight respiratory illnesses and symptoms investigated at two successive annual examinations of 10,106 white children living in six cities. Paternal smoking was associated with smaller but still substantial increases. Illness and symptom rates were linearly related to the number of cigarettes smoked by the child's mother, and were higher for children of current smokers than for children of ex-smokers. The associations between maternal smoking status and childhood respiratory illnesses and symptoms were reduced but not eliminated by adjustment for parental illness history. Levels of forced expiratory volume in 1 sec (FEV$_1$) were significantly lower for children of current smokers than for children of nonsmokers at both examinations, and highest for children of ex-smokers. Levels of forced vital capacity (FVC) were lower for children of nonsmokers than for children of current smokers at both examinations, but the difference was statistically significant only at the first examination. Both the increase in mean FVC and the decrease in mean FEV$_1$ among children of current smokers were linearly related to daily cigarette consumption. None of the respiratory illnesses and symptoms studied was significantly associated with exposure to gas cooking in the child's home. Results provide strong support for a causal effect of sidestream cigarette smoke on increased respiratory illness and reduced FEV$_1$ values in preadolescent children. (Auth. Abs. Mod.)

CARDIOVASCULAR DISEASES

84–1138. Balaz, V. **Rizikove Faktory Vyzivy a Fajceni v Epidemiologii Aterosklerozy u Bratislavskych 45 az 54 Rocnych Muzov a Zien. [Nutritional and Smoking Hazard Factors in the Epidemiology of Atherosclerosis in 45 to 54 Years Old Males and Females From Bratislava.]** *Lekarsky Obzor* 31(8): 425-432, August 1982, Polish.

Smoking is cited as a risk factor for atherosclerosis, but a detailed analysis is not provided in this study of approximately 800 men and 800 women, aged 45 to 54 years, in Bratislava, Czechoslovakia. However, it is emphasized that smoking potentiates and aggravates other risk factors, in addition to its own unique effects. Subjects were surveyed for total percentage of body fat, serum cholesterol and lipid levels, manual vs. sedentary occupation, smoking habits, age, gender, and body weight. Approximately 60 percent of the men across all occupations smoked >10 cigarettes/day, compared with 16 to 17 percent of the women. The greatest incidence of known risk factors occurred in white-collar males, with elevated levels of triglycerides found in 38.4 percent, elevated lipoproteins in 48.5 percent, latent obesity in 29.8 percent, and hypertension in 35.6 percent of those studied. The lowest incidence of risk factors was seen in white-collar females, who were also noted to have the best overall dietary habits. Clinical obesity and elevated serum cholesterol were most frequent in blue-collar females at 29.2 and 25.6 percent, respectively.

84–1139. Bjartveit, K.; Foss, O. P.; Gjervig, T. **The Cardiovascular Disease Study in Norwegian Counties. Results From First Screening.** *Acta Medica Scandinavica* Supplement 675: 1-184, March 1983.

Results of the first screening in a cardiovascular disease study, started in 1974, includes analysis of smoking prevalence and habits of the 57,859 participants (88 percent response in the three counties covered). It was hoped that results would show contrasts between the counties to explain the marked county differences in cardiovascular disease mortality. Mortality is highest in Finnmark, Norway's northernmost county, and lowest in Sogn og Fjordane and Oppland, both located in southern Norway. Data on smoking habits were complete for all but 434 subjects, and showed the highest prevalence for both men and women in all age categories in Finnmark (over 60 percent of men and about 50 percent of women are daily smokers). Detailed information by age, sex, county, and smoking-related symptoms and diseases is provided. Differences between the healthy population and persons reporting various diseases and symptoms are used to derive risk factor profiles characteristic of these subgroups. Information includes (1) cardiovascular disease and symptom prevalence (main group distribution); (2) serum lipid concentrations; (3) blood pressure, treatment of hypertension, and stroke prevalence; (4) risk scores; (5) serum glucose and diabetes prevalence; (6) physical activity; (7) weight and height; (8) familial factors; and (9) a general discussion and final remarks. Additional information on smoking habits is provided in 84-1092.

84–1140. Cambien, F.; Ducimetiere, P.; Richard, J.-L.; Jacqueson, A.; Guillanneuf, M. **Alcohol Consumption and the Duration of Systolic Time Intervals in Middle-Aged Men.** *Journal of Studies on Alcohol* 44(4): 591-599, July 1983.

Results of a study, designed to correlate the current and lifetime alcohol consumption of 149 Parisian civil servants (all males, mean age 50.4) with systolic time intervals, showed no increase in mean preejection period (PEP) and mean ratio of PEP to left ventricular ejection time (LVET) with cigarette smoking, although the PEP and PEP/LVET ratio were elevated in the same professional group 2 years ago. Of interest is the finding that the mean number of cigarettes smoked/day dropped from 8.9±9.7 in the earlier study to 4.9±8.6 at present. After controlling for cigarette consumption, age, and individual coronary variables, total lifetime alcohol consumption had a significant negative association with PEP and PEP/LVET ratio. However, alcohol consumption was positively (but insignificantly) associated with LVET after controlling for heart rate, which was highly negatively associated with LVET. Cigarette consumption was not independently associated with systolic time intervals. Results suggest a moderate progressive increase in myocardial contractility with an increase in alcohol consumption. The association between PEP and PEP/LVET ratio and alcohol consumption is independent of age, heart rate, blood pressure, ponderal index, and cigarette consumption.

84–1141. Castelli, W. P. **Epidemiology of Coronary Heart Disease: The Framingham Study.** *American Journal of Medicine* 76(2A): 4-12, February 27, 1984.

Results from the first 14 years of the Framingham study indicate that cigarette smokers and other smokers who inhale have about 1.5 times the risk of coronary heart disease (CHD) compared with nonsmokers. Men who smoke filter cigarettes are at higher risk of CHD than nonfiltered cigarette smokers: for men under age 55, myocardial infarct incidence (per 1,000) was 0.40 for filtered cigarette smokers vs. 0.024 for users of nonfiltered products, and for men over 55, 0.032 vs. 0.020. One year after cessation of smoking, risk of myocardial infarct returned to the nonsmokers' level. While CHD is a multifactorial disease, lipid abnormalities are the most fundamental factors in the basic process. It is stressed that isolated management of one risk factor (e.g., hypertension) is poor medicine; all major factors must be addressed if CHD is to be significantly reduced.

84–1142. Cox, R. H.; Tulenko, T.; Santamore, W. P. **Effects of Chronic Cigarette Smoking on Canine Arteries.** *American Journal of Physiology* 246(1, Part 2): H97-H103, January 1984.

Results show that some significant changes in arterial wall properties occurred in beagle dogs exposed to chronic cigarette smoke. Segments of carotid and femoral arteries were obtained from 9 beagles subjected to cigarette smoking (12 cigarettes/day for 2 years) and from 10 unexposed controls. Segments were used for in vitro studies of active and passive mechanics, dose—responses to norepinephrine (NE) and K^+, connective tissue content, and water and electrolyte content and distribution. No significant differences in passive arterial wall mechanics or geometry at 100 mm Hg were found, but a small (significant) increase in passive stiffness was found at wall loads corresponding to higher values of pressure for arteries from smokers. No significant changes in connective tissue composition were found at either site. No significant differences in maximum active force development were found at either site. However, values of active force near the middle of the active force—length curve were lower ($p<0.05$) for smoker's arteries when expressed as a fraction of the maximum developed force. No differences in cell volume were found in arteries from the two groups. Mg^{2+} and K^+ content of the femoral and Ca^{2+} content of both arteries were greater in smokers. No difference in NE dose—response relations were found for either site. A statistically significant decrease in responsiveness to K^+ was found for femoral arteries from smokers, but not for the carotids. (Auth. Abs. Mod.)

84–1143. Hataway, J.; Oberman, A. **Prevention of Coronary Disease: An Update.** *Alabama Journal of Medical Sciences* 21(1): 58-67, January 1984.

Cigarette smoking, hypertension, and hypercholesterolemia double the risk of coronary heart diseases (CHD), the leading cause of death in the United States. CHD is a catastrophic and frequently silent disease that increases the risk of further cardiovascular complications by 5 to 20 times. CHD is based on the interaction of multiple familial and environmental factors, but environmental factors predominate in disease development and progression. Other factors that may influence progression are physical inactivity, obesity, clinical diabetes, and type A personality. Two prevention approaches are mass intervention in the general population or intervention only for individuals at high risk. However, if only individuals at high risk are given intervention, the larger number of individuals with less absolute risk (accounting for the majority of excess morbidity and mortality) will go untreated. Animal models and limited arteriographic studies among humans clearly demonstrate the possibilities for regression of atherosclerosis. Clinical trials involving treatment of mild, moderate, and severe hypertension indicate significant reduction of major cardiovascular complications. Evidence shows that the risk associated with cigarette smoking is clearly reduced upon cessation of smoking, but is not reduced with usage of low-tar cigarettes. The role of hypercholesterolemia, the most controversial major risk factor, should be resolved by an ongoing definitive trial to substantiate the value for lowering cholesterol to reduce risk. Substantial decreases in the level of risk depend on alteration of multiple factors among large groups, rather than drastic reduction of isolated risk factors in a few individuals. If current recommendations for lowering risk were achieved, as much as a 63 percent reduction in the incidence of coronary heart disease might be expected. (Auth. Abs. Mod.)

84–1144. Kannel, W. B.; Schatzkin, A. **Risk Factor Analysis.** *Progress in Cardiovascular Diseases* 26(4): 309-332, January-February 1984.

Smoking is included as one of the main risk factors for coronary heart disease (CHD) in a detailed analysis of the risk factors, the potential for reducing CHD risk by risk factor modification, and the effect of changes in risk factor prevalence over the last few years on CHD mortality. Among the main factors discussed are serum cholesterol, blood pressure, carbohydrate tolerance, and certain ECG abnormalities; all have shown an independent relationship with CHD, which is clearly a multifactorial process. Smoking is one of the many risk factors that are potentially modifiable, since data have tended to indicate an independent, transient, noncumulative, reversible triggering effect from smoking. The effect is more closely related to the number of cigarettes smoked/day than to duration of habit, and those who quit have only half the risk of myocardial infarction (MI) compared with those who continue to smoke. Smoking appears to be a higher risk in persons with arterial problems, showing an association with risk of MI independent of the levels of other cardiovascular risk factors. Although the relative risk is greater for smoking and development of cancer, the attributable risk is higher for CHD; thus, the elimination of smoking would actually save more lives from CHD than from lung cancer, since so many more people contract CHD. While the recent decrease in deaths from CHD and accelerated decline in general cardiovascular mortality since 1968 may be attributed to advances in medicine, preventive measures, and changes in lifestyle, trends in smoking, particularly in middle-aged men, may have contributed to the decline. One unequivocal conclusion is that these diseases are not an inevitable consequence of aging or genetic makeup, and therefore can be prevented. However, effective risk factor control will require development of better skills for behavior modification, and also intensive health education, public health measures, and preventive medicine.

84–1145. Lang, T.; Degoulet, P.; Aime, F.; Fouriaud, C.; Jacquinet-Salord, M.-C.; Laprugne, J.; Main, J.; Oeconomos, J.; Phalente, J.; Prades, A. **Relation Between Coffee Drinking and Blood Pressure: Analysis of 6,321 Subjects in the Paris Region.** *American Journal of Cardiology* 52: 1238-1242, December 1, 1983.

As part of an investigation on the possible link between coffee drinking and high blood pressure, the effect of coffee consumption was studied in subgroups of smokers and nonsmokers. Age-adjusted mean levels of blood pressure (BP) were established in 3,524 nonsmokers and 2,797 smokers, and systolic, but not diastolic, BP levels were significantly higher in smokers compared with nonsmokers. Among nonsmokers, both age-adjusted systolic and diastolic BP increased with amount of coffee consumed, while only adjusted systolic BP increased significantly with coffee consumption among smokers. Findings emphasize the complexity of a possible link between coffee drinking and coronary heart disease (CHD) and may explain the contradictory results of epidemiologic studies. Coffee consumption may raise BP and cholesterol level and, hence, increase the risk of CHD, but its close association with cigarette smoking may modify the effect of caffeine on BP, low density lipoproteins, and total cholesterol.

84–1146. Limet, R. **Tabac et Maladies Cardio-Vasculaires. [Tobacco and Cardiovascular Diseases.]** *Revue Medicale de Liege* 38(17): 652-655, September 1, 1983, French.

Cigarette consumption and related nicotine and carbon monoxide effects increase the incidence and severity of diseases of the coronary system and peripheral arteries, and significantly alter the survival curve of coronary disease patients who continue to smoke. While it is desirable, from a public health point of view, to limit tobacco use, it is essential to strictly forbid smoking among patients suffering from angina or intermittent claudication. Epidemiologic data indicate that regular use of tobacco increases occurrence of myocardial infarctions, mortality from blocked arteries, and the proportion of sudden death among persons with heart disease. Nicotine increases pressure in the arteries, which affects oxygen availability to the patient, and smoking further affects the peripheral arteries, especially in the lower extremities. While arterial hypertension, cigarette smoking, and hypercholesterolemia are individual risk factors for vascular accident, a combination of these factors greatly multiplies risk. Lower incidence of coronary disease in Yugoslavia and Japan, where tobacco consumption is very high, may be related to cholesterol levels, which are less than 2 g/1. Use of tobacco aggravates atherogenesis; one study indicated a fivefold increase in men aged 40 to 49. Data have shown that cessation of smoking can reverse various adverse effects.

84–1147. Lowry, P. J.; Glover, D. R.; Mace, P.J.E.; Littler, W. A. **The Pattern and Severity of Coronary Artery Disease in Asians and Whites Living in Birmingham.** *Postgraduate Medical Journal* 59(696): 634-635, October 1983.

Racial factors affecting the incidence of and risk factors for coronary artery disease (CAD) were examined in a sample of Asian and Anglo smokers and nonsmokers. Each Asian patient with CAD (n=34) was matched for age, sex, blood pressure, and duration of symptoms with two white patients with CAD. Blind assessment of coronary arteriograms demonstrated significantly more severe disease in Asians, although the distribution of disease was similar in both groups. The Asian group had significantly fewer smokers (68 percent compared with 91 percent of whites), lower cholesterol levels, and weighed less than whites. (Auth. Abs. Mod.)

84–1148. Lyon, J. L.; Schuman, K. L.; Avlon, E. D.; Langford, H. G.; Nicholson, J. P.; Teichman, S. L.; Alderman, M. H.; Sos, T. A.; Pickering, T. G.; Laragh, J. H. **Cigarette Smoking in Renovascular Hypertension. (Letters).** *Lancet* 1(8368): 104-105, January 14, 1984.

Data reported earlier (*Lancet* 2(8353): 765-766, October 1, 1983) on the highly significant association between cigarette smoking and renal artery stenosis is questioned, citing conceptual and methodological faults in the study, particularly in establishing exact smoking habits, and ignoring other confounding variables such as sex, diabetes, and hypertension. The designation as a retrospective cohort study rather than a case—control study is also considered wrong, leading to errors in measurement of risk. The original authors respond that the study designation is only a question of semantics, and the additional analysis provided adds support to the original observations. Even when patients whose smoking histories were obtained only by telephone are eliminated, a twofold difference in risk still exists. In answer to a separate question on the ratio of blacks and whites, the authors note that while the number of blacks was small, the relationship between smoking and renal artery stenosis was still apparent. See also 84-0512.

84–1149. Maouad, J.; Fernandez, F.; Barrillon, A.; Gerbaux, A.; Gay, J. **Diffuse or Segmental Narrowing (Spasm) of the Coronary Arteries During Smoking Demonstrated on Angiography.** *American Journal of Cardiology* 53(2): 354-355, January 15, 1984.

The vasoconstrictive effect of cigarette smoking on the coronary arteries was demonstrated in five angina patients (four smoked ≥ 10/day). Each patient was given one cigarette to smoke, and an angiogram was performed after half the cigarette had been consumed. Smoking by the patients resulted in (1) a localized spasm with a noticeable diffuse reduction in the lumen of the left anterior descending artery (LAD) in patients 1 and 3; (2) an increase in the narrowing of the lumen of the LAD from 20 to 40 percent in patient 5; (3) a global 20 percent decrease of the lumen in patient 2; and (4) a triggering of angina and a 45 percent global decrease of the lumen of the LAD in patient 4, a nonsmoker.

84–1150. Marshall, M. **Angiopathierisikofaktor Inhalierendes Rauchen. [Smoking With Inhalation as a Risk Factor of Angiopathy.]** *Zeitschrift fur Allgemeinmedizin* 60(4): 142-150, February 10, 1984, German.

Numerous animal and human studies have shown that even low carbon monoxide (CO) concentrations, with a corresponding 3 to 5 percent increase in carboxyhemoglobin (COHb), cause functional damage to the cardiovascular system. Apparently, CO exposure (such as occurs in cigarette smoking) can lead quickly to additional morphologically significant damage. At the same time, nicotine produces acute functional circulatory and hemorrheological effects. CO from cigarette smoke must therefore be considered a potent factor in damage to the vascular system. Hence, a "less harmful cigarette" must also produce a low CO content in smoke. Regarding the problem of "passive smoking," it appears that, under normal environmental conditions, the effects of CO and nicotine are not significant in healthy people. Thus far, no reliable data are available on the significance of other components of cigarette smoke in cardiovascular disease risk.

84–1151. Puska, P.; Salonen, J. T.; Nissinen, A.; Tuomilehto, J.; Vartiainen, E.; Korhonen, H.; Tanskanen, A.; Ronnqvist, P.; Koskela, K.; Huttunen, J. **Change in Risk Factors for Coronary Heart Disease During 10 Years of a Community Intervention Programme (North Karelia Project).** *British Medical Journal* 287(6408): 1840-1844, December 17, 1983.

Data show that smoking among middle-aged males declined by 28 percent ($p<0.001$); among females, there was a nonsignificant reduction of 14 percent. Reductions in smoking, serum cholesterol concentrations, and blood pressure were among the central intermediate objectives of the North Karelia Project for cardiovascular disease risk reduction. The effect of the program over a 10-year period was evaluated by examining independent random population samples at the outset (1972) and 5 (1977) and 10 (1982) years later both in the program and in a matched reference area. Over 10,000 subjects were studied in 1972 and 1977 (participation rate about 90 percent), and roughly 8,000 subjects in 1982 (participation rate about 80 percent). Analyses were conducted of the estimated effect of the program on the risk factor population means by comparing the baseline and 5-year and 10-year followup results in the age range from 30 to 59 years. Other effects of the program (net reduction in North Karelia) at 10 years among the middle-aged male population included a 3 percent reduction in mean serum cholesterol concentration ($p<0.001$), a 3 percent fall in mean systolic blood pressure ($p<0.001$), and a 1 percent fall in mean diastolic blood pressure ($p<0.05$). Among females the reductions were, respectively, 1 percent (nonsignificant), 5 percent ($p<0.001$), and 2 percent ($p<0.05$). (Auth. Abs. Mod.)

84–1152. Renaud, S. **Risk Factors for Coronary Heart Disease and Platelet Functions.** *Advances in Experimental Medicine and Biology. Thrombosis and Cardiovascular Diseases.* 164: 129-144, 1980.

Farmers in West Scotland, where the coronary heart disease (CHD) mortality, saturated fat intake, and platelet reactivity are very high, were shown to have increased platelet reactivity (15 to 100 percent, depending on the test) after smoking one cigarette. In a related study, platelet functions, blood lipids, and saturated fat intake were compared in male farmers from two regions (Moselle and Var) in France. In Moselle, saturated fat intake is 16 percent of diet, similar to that in North America and West Scotland; in Var it is 12 percent, approximately that of Italy. CHD mortality is very high in Moselle and low in Var. No differences were found in total cholesterol, HDL-cholesterol, or triglycerides, but coagulation and platelet clotting activity were markedly accelerated in the Moselle group. One year after fat intake was lowered to 12 percent in the Moselle group, both platelet functions were drastically reduced. Platelet function appears to be the main intermediate link between most environmental risk (and preventive) factors and CHD.

84–1153. Ring, T.; Kristensen, S. D.; Jensen, P. N.; Mourits-Andersen, T.; Madsen, H.; Dyerberg, J. **Cigarette Smoking Shortens the Bleeding Time.** *Thrombosis Research* 32(6): 531-536, December 15, 1983.

Cutaneous bleeding time was shortened after high-nicotine cigarettes were smoked. Measurements were taken in 10 healthy males, who were accustomed to smoking high-nicotine cigarettes, before and immediately after they smoked either two high-nicotine cigarettes (Cecil, 2.4 mg nicotine) or two nicotine-free cigarettes (Free) within 15 minutes. Subjects had been abstinent from alcohol for 12 hours and coffee and smoking (although five had slight passive exposure) for 8 hours. The procedure was repeated 1 to 3 days later in a crossover experiment with randomized sequence of smoking. While bleeding time was normal in all subjects, it was shortened after smoking Cecil in all but two (in whom it was unchanged), and was unchanged after Free cigarettes were smoked. Results showed that adenosine-5-diphosphate-induced primary platelet aggregation was not enhanced, and the number of circulating platelet aggregates did not change due to smoking either cigarette type.

84–1154. Russell, R. O., Jr. **Protection of Ischemic Myocardium During or After Convalescence From Acute Myocardial Infarction.** *Chest* 85(2): 248-252, February 1984.

Results from two previous studies (in Norway and Sweden) are cited as evidence that cessation of smoking reduces the risk of myocardial infarct, the risk of reinfarction, and the mortality risk associated with coronary heart disease in middle-aged males. In Sweden, a 50 percent reduction in cardiovascular deaths was reported among those who had quit smoking. Other aspects of the problem of identifying the patient at high risk of myocardial infarction or reinfarction are discussed, and treatment procedures are reviewed, including (1) the use of beta-blocking agents to protect ischemic myocardium; (2) the role of diet in reducing serum cholesterol and triglycerides; (3) the value of nitrates, anticoagulants, and antiarrhythmic agents; and (4) indications for coronary artery bypass surgery.

84–1155. Santos, M. T.; Valles, J.; Aznar, J.; Beltran, M.; Herraiz, M. **Effect of Smoking on Plasma and Platelet Fatty Acid Composition in Middle-Aged Men.** *Atherosclerosis* 50(1): 53-62, January 1984.

Studies on the effect of smoking (in 173 men aged 40 to 62) on plasma and platelet fatty acid composition and plasma malondialdehyde-like material (MDA-LM) revealed an increase of saturated fatty acids and a concomitant decrease in unsaturated fatty acids in subjects who smoked >20 cigarettes/day, compared with nonsmokers. In platelets, an increase in myristic acid (14:0) and palmitoleic acid (16:1) was found. Evaluation of platelet lipid fractions showed an increase of 14:0 and 16:0 + 16:1 in phospholipids. The plasma MDA-LM level was significantly higher in subjects who smoked >10 cigarettes/day. These biochemical variations may be related to the increased incidence of coronary heart disease in smokers. (Auth. Abs. Mod.)

84–1156. Schenk, K. E. **Fruhrehabilitation in Form der Anschlussheilbehandlung bei Patienten mit Herz-Kreislauf-Krankheiten Sowie Kardialen Operationen. [Early Rehabilitation in the Form of Continuous Treatment of Patients With Heart and Circulatory Diseases as Well as Heart Surgery.]** *Fortschritte der Medizin* 101(40): 1819-1822, October 1983, German.

Among 231 patients undergoing rehabilitation after heart attacks or heart surgery, 70 percent were smokers on admission to the rehabilitation clinic, but only 7 percent were still smoking after 4 to 6 weeks of treatment. Behavioral modification included suggestions on smoking cessation, dietary changes, and exercise. After treatment, the number of patients with high blood pressure dropped from 17 percent to 0 percent; mean blood cholesterol was reduced from 311 to 238 mg percent, triglycerides from 240 to 180 mg percent, and weight loss averaged 3.5 kg. After training, 92 percent returned to work, compared with 50 percent reported in other studies.

84–1157. Schmidt, F. **Rauchen und Herzinfarkt. [Smoking and Myocardial Infarction.]** *Zeitschrift fur Allgemeinmedizin* 60(4): 151-155, February 10, 1984, German.

A brief review of retrospective and prospective studies concludes that smoking is a primary risk factor of myocardial infarction (MI), and the most important single risk factor for MI before the age of 45. The ratio of risk for smokers to that for nonsmokers is 15 to 1 in the <45 age bracket. Also, the incidence of MI in ex-smokers is clearly lower than in smokers, but higher than in nonsmokers, showing that abstaining from smoking is advantageous for MI patients. It is recommended that physicians stop smoking as an example for patients.

84–1158. Stamler, J.; Stamler, R. **Intervention for the Prevention and Control of Hypertension and Atherosclerotic Diseases: United States and International Experience.** *American Journal of Medicine* 76(2A): 13-36, February 27, 1984.

While no unifactorial randomized controlled prevention trials have been done with smoking, the Multiple Risk Factor Intervention Trial (MRFIT) assessed primary prevention of fatal coronary heart disease (CHD) with regard to hypercholesterolemia, hypertension, and smoking. Cessation of smoking was inversely proportional to the number of cigarettes smoked/day at baseline. Among normotensive smokers, considerably more men receiving special intervention stopped smoking and the intervention group had 48.7 percent less 7-year CHD mortality compared with men receiving usual care. The United States population as a whole has a large high-risk segment for CHD. Since the late 1950s, significant population-wide changes have occurred in lifestyles (smoking, diet, exercise habits), especially among the better educated. The proportion of persons with detected, treated, and controlled high blood pressure has risen markedly in all population strata. Consequently, a favorable shift has occurred in the population distribution of the major established risk factors: cigarette smoking, "rich" diet, hypercholesterolemia, and high blood pressure. It is inferred that the steady and marked declines in death rates in the United States from coronary heart disease, stroke, all cardiovascular diseases, and all causes since 1968 are related to reductions in these risk factors. (Auth. Abs. Mod.)

84–1159. Taylor, S. H. **Oxprenolol in Clinical Practice.** *American Journal of Cardiology* 52(9): 34D-42D, November 10, 1983.

In a review of oxprenolol's therapeutic role in controlling cardiovascular disease, it is noted that the drug suppresses the cardiovascular and metabolic effects of smoking. Cigarette smoking is associated with marked sympathoadrenal stimulation due to the nicotine-induced release of catecholamines from the adrenal medulla. In normal subjects and in patients with ischemic heart disease, oxprenolol reduced the catecholamine stimulation of the heart and also the associated rise in plasma concentration of free fatty acids induced by cigarette smoking. Primary prevention of coronary heart disease (CHD) in hypertensives and secondary prevention of CHD in myocardial infarct patients with oxprenolol is discussed. The efficacy of oxprenolol in the treatment of angina pectoris, arrhythmias, hypertension, and minor cardiovascular symptoms is also evaluated. (Auth. Abs. Mod.)

84–1160. U.S. Department of Health and Human Services. **The Health Consequences of Smoking: Cardiovascular Disease. A Report of the Surgeon General.** U.S. Department of Health and Human Services, Public Health Service, Office on Smoking and Health, Publication No. DHHS(PHS) 84-50204, 1983, 395 pp.

The 15th report on the health consequences of smoking, by the Department of Health and Human Services, provides an in-depth review of the relationship between tobacco use and cardiovascular disease. The eight sections of the report are (1) introduction and overview; (2) arteriosclerosis; (3) coronary heart disease (CHD); (4) cerebrovascular disease; (5) atherosclerotic peripheral vascular disease and aortic aneurysm; (6) pharmacological and toxicological implications of smoke constituents on cardiovascular disease; (7) changes in cigarette smoking behavior in clinical and community trials; and (8) the effect of smoking cessation on coronary heart disease. Two appendices provide data on trends in cardiovascular disease and on trends in U.S. cigarette use from 1965 to 1980. The report concludes that cigarette smoking is one of the three major independent coronary heart disease risk factors, the others being hypertension and elevated serum cholesterol. Because of the higher prevalence of smoking, however, it ranks as the largest preventable cause of coronary heart disease death in the United States. Smoking also acts synergistically with other major risk factors to greatly increase CHD risk, and contributes to the development of atherosclerotic lesions, which are the predominant underlying cause of cardiovascular disease. Cessation of smoking is the single most important intervention in the management of vascular disorders.

84–1161. Wilhelmsen, L. **Risk Factors for Coronary Heart Disease in Perspective. European Intervention Trials.** *American Journal of Medicine* 76(2A): 37-40, February 27, 1984.

Results of two major intervention trials aimed at reducing the incidence of coronary heart disease (CHD) and stroke are summarized. In the 5-year Oslo trial of diet and smoking intervention, 1,232 healthy normotensive males at high risk of CHD were studied. In the intervention group, (1) mean tobacco consumption was reduced 45 percent over controls; (2) 25 percent quit smoking, compared with 17 percent of controls; and (3) myocardial infarct and sudden death were 47 percent less than controls. In the World Health Organization (WHO) Multifactorial Trial, several populations were studied. Among the English group, 18,200 male factory workers received intervention in diet, smoking, and exercise. Self-reported cigarette smoking in the intervention group was moderately reduced; other changes were minor. Among the Belgian group of 19,409 males (20 percent at high risk of CHD), there was a 15.8 percent reduction in combined risk factors. Other centers in the WHO trial have reported varying results.

84–1162. Woodson, P. P.; Buzzi, R.; Baettig, K. **Effects of Smoking on Vegetative Reactivity to Noise.** *Sozial- und Praventivmedizin* 28: 240-241, May 1983.

Real smoking had a pronounced dampening effect on subjective annoyance to noise, which occurred even before subjects were allowed to smoke, suggesting tranquilization by anticipation. Sixty adult women were assigned to one of five treatment groups: (1) smoking-deprived subjects who smoked; or (2) sham-smoked; (3) nonsmokers who sham-smoked; or (4) nondeprived smokers who smoked; or (5) sham-smoked. During the three phases of the test (noise exposure, three 36-sec bursts; smoking or sham-smoking; and noise exposure, five 36-sec bursts), subjective annoyance to each noise burst was recorded, blood pressure determined, and cigarette puffing profile measured (breath carbon monoxide as index of smoke inhalation). The interactions between noise and smoking-induced sympathetic arousal varied for different recording sites. The importance of cardiovascular cues in the calming effect of smoking should therefore be questioned, and the role of anticipation as a mediating variable should be explored.

See also **84–1018, 84–1092**

PREGNANCY AND INFANT HEALTH

84–1163. Agrawal, P.; Chansoriya, M.; Kaul, K. K. **Effect of Tobacco Chewing by Mothers on Placental Morphology.** *Indian Pediatrics* 20(8): 561-565, August 1983.

Data from 96 sets of mothers, their babies, and the placentas showed significant differences between the birth weight of 48 infants of mothers who used tobacco compared with 48 infants of mothers who were nonusers. Women were matched for age, socioeconomic status, literacy, parity, birth interval, height, and weight, and gestational age and sex of the baby. The birth weight, length, and circumference of the skull were recorded, and placental studies were conducted. Placentas of tobacco consumers were found to be an average of 65.9 g heavier. Mothers who had consumed tobacco regularly for 6 years or more in the form of tobacco leaves and lime had significantly heavier placentas. Tobacco chewing provides a situation where the pharmacologic consequences of the ingredients in tobacco are not influenced by carbon monoxide. However, similar results (higher placental weight, lower birth weight) have also been found for mothers who smoke. How chewing tobacco influences fetal and placental outcome remains only conjecture. (Auth. Abs. Mod.)

84–1164. Aro, T. **Maternal Diseases, Alcohol Consumption and Smoking During Pregnancy Associated With Reduction Limb Defects.** *Early Human Development* 9(1): 49-57, December 1983.

While medication, fever, and/or other factors may explain the observed association of reduction limb defects (RLD) with maternal influenza, the relationship of RLD and maternal smoking was not explained by alcohol consumption or mother's age. Among 453 confirmed RLD cases, 124 had additional defects of various organs. The risk of RLD in children of mothers who smoked >5 cigarettes/day was increased in the total group, and significantly higher in the group with no additional malformations (age- and alcohol use-adjusted odds ratio 1.7). No significant interaction between alcohol consumption and smoking was found, although alcohol was statistically associated with cases with no additional defects, but not with cases with additional defects.

84–1165. Bassi, J. A.; Rosso, P.; Moessinger, A. C.; Blanc, W. A.; James, L. S. **Fetal Growth Retardation Due to Maternal Tobacco Smoke Exposure in the Rat.** *Pediatric Research* 18(2): 127-130, February 1984.

A model of maternal smoking during pregnancy was applied to three groups of rats: control, pair-fed, and smoke-exposed (continuous smoke exposure for cycles of 7 min, 16 times/day from days 5-20 of gestation). On day 21, all fetuses were removed, and fetal brain, liver, lungs, and placentas were weighed and analyzed for nucleic acid. Significant differences in body weight were noted for smoke-exposed, pair-fed, and control groups (4.60 g, 5.21 g, and 5.85 g, respectively). Neither fetal brain weight nor nucleic acid content were affected by maternal exposure to smoke, whereas fetal liver and lung weight were significantly decreased (346 mg compared with 417 mg and 491 mg for pair-fed and control groups, respectively). RNA and DNA content in liver and lungs of smoke-exposed animals was also significantly decreased compared with pair-fed and control groups. Results indicate that fetal growth retardation associated with maternal exposure to tobacco smoke in the rat corresponds to a disproportionate type, and occurs without placental growth retardation, similar to the effects of uterine artery ligation in the rat. (Auth. Abs. Mod.)

84–1166. Bewley, B. R. **Smoking in Pregnancy.** *British Medical Journal* 288(6415): 424-426, February 11, 1984.

While the proportion of heavy smokers (≥20 cigarettes/day) among British men decreased from 1972 to 1982, a similar change in female heavy smokers (who are mostly in social class V) has not been noted. A review of the complex consequences of this differential smoking cessation pattern to both women smokers and their children indicates (1) an association between maternal smoking and reduced birth weight resulting from retarded intrauterine growth, and attributes chronic fetal oxygen deficiency to nicotine- and carboxyhemoglobin-related vasoconstriction; (2) both maternal and paternal smoking adversely affect infants' respiratory health, and more children of smokers have bronchitis and pneumonia during the first year of life; and (3) a pregnant heavy smoker is more than twice as likely as a nonsmoker to lose her baby at or near birth. More mothers who quit smoking during early pregnancy admitted to knowledge of possible hazards to the fetus than those who smoked throughout pregnancy, and the proportions admitting such knowledge were higher among those receiving medical advice. Cigarette advertising specifically aimed at young women has contributed to the smoking epidemic, which, though clearly declining, has been complicated by the issues of women's emancipation and smokers' freedom, and by tobacco companies pushing their dangerous drug.

84–1167. Foidart, J. M. **Tabac et Grossesse. [Tobacco and Pregnancy.]** *Revue Medicale de Liege* 38(17): 655-663, September 1, 1983, French.

A review of the literature shows that it is difficult to determine the specific effect of smoking on pregnancy, since smoking is only one of many factors that may influence outcome, such as ethnic, genetic, and biological background and socioeconomic, nutritional, and environmental factors. Newborns of smoking mothers do weigh an average of 200 g less than infants of mothers who do not smoke. Therefore, pregnant women should be advised not to smoke, since smoking also has harmful effects on the respiratory and vascular systems. For those who find it difficult to quit, reducing consumption to 4 or 5 cigarettes/day would reduce the smoking-associated risks and enhance the chances for a good pregnancy. Besides lower birth weight, smoking appears to be a factor in the early detachment of the placenta and of increased occurrence of placenta previa. Other possible outcomes that may be influenced by smoking include spontaneous abortions, lower maternal weight gain, premature births, difficulties in delivery, congenital malformations, and infant mortality. The long-term effects on children born to mothers who smoked during pregnancy are still under study.

84–1168. Hickner, J.; Westenberg, C.; Dittenbir, M. **Effect of Pregnancy on Smoking Behavior: A Baseline Study.** *Journal of Family Practice* 18(2): 241-244, February 1984.

Results of a retrospective family practice survey, designed to discover if there is a long-lasting effect of smoking cessation during pregnancy, are presented. Self-administered questionnaires were completed by 66 women (all smokers prior to most recent pregnancy) in three family practices. Women were questioned regarding the number of cigarettes smoked before and during pregnancy and about current smoking level. Twelve women (18 percent) stopped smoking during pregnancy, but only seven (11 percent) were still abstinent at the time of the survey. Smoking had been reduced by 50 percent in 28 women (42 percent) during pregnancy, but only 18 had maintained the reduction 6 months to 5 years after delivery. Further study is needed to determine if intensive antismoking efforts during routine prenatal care would be more effective and long lasting. (Auth. Abs. Mod.)

84–1169. McCredie, J.; Kricker, A.; Elliott, J.; Forrest, J. **Congenital Limb Defects and the Pill. (Letter).** *Lancet* 2(8350): 623, September 10, 1983.

Further evidence is presented on the relationship of oral contraceptive (OC) use during pregnancy and limb defects in newborns, noting that the relative risk (RR) remained high even after allowance for smoking. A case—control study of 155 liveborn children (two controls/case) found that the RR for 18 mothers who took OCs during early pregnancy was 23.9 (23.1 when adjusted for smoking). Findings support earlier studies on the relationship of OCs with limb deformities, but do not support a sex-specific effect. The rarity of the clinical occurrence of deformities suggests that further studies are needed on the pathogenesis of congenital limb deformities.

84–1170. Menghetti, E. **Recenti Acquisizioni Sulle Conseguenze nei Bambini del Fumo Materno in Gravidanza. [Maternal Smoking in Pregnancy: Recent Acquisitions on Its Consequences in Infants.]** *Rivista Italiana di Pediatria* 9(2): 111-112, March-April 1983, Italian.

Research indicates maternal smoking during pregnancy can affect the outcome in several ways: lower birth weight of infants, smaller size, delays in learning to cry and suckle, difficulties in adapting to breast feeding, lower Apgar indices, and higher hemoglobin and hematocrit. Data on 195 newborns demonstrated that infants born to mothers who smoke also had smaller cranial circumference. In children of mothers who smoked, who were followed from the age of 28 days to 5 years, there was a higher rate of mortality, longer and more frequent need for hospitalization, and a higher percentage of respiratory ailments. Other studies have shown such children are slower in learning to read and other learning skills, show lower mental capabilities, and are smaller in stature. It is noted that since tobacco is known to contain more than 4,000 chemical components, it is imperative that women be made aware of the dangers of smoking, particularly during pregnancy.

84–1171. Moggian, G.; Parmeggiani, A.; Forcucci-Zulli, M.; Tumedei, U.; Zulli, P. **Indagine Statistica Sugli Effetti del Fumo in Gravidanza. [Statistical Investigation of the Effects of Smoking During Pregnancy.]** *Giornale Italiano di Ostetricia e Ginecologia* 5(11): 1285-1302, November 1983, Italian.

In a retrospective study of smoking effects during pregnancy, mothers who smoked had higher miscarriage rates, more premature infants, and lower birthweight babies compared with nonsmokers. Of 1,595 mothers surveyed, 918 were nonsmokers, 610 habitual smokers, 25 occasional smokers, and 42 ex-smokers. Fetal loss was higher among smokers at each of the first 5 months, particularly the first month (2.4 percent in smokers; 0.9 percent in nonsmokers). Among first-born infants, nonsmoking mothers had 9.62 percent premature births, those who quit smoking during pregnancy 12.5 percent, those who reduced smoking during pregnancy 16.6 percent, and current smokers 19.44 percent. Among second-born infants, figures were 7.39, 15.97, 19.05, and 20 percent, respectively. No significant differences were found for rates of live births and for state of health of the newborn.

84–1172. Ounsted, M. K.; Moar, V. A.; Scott, A. **Small-for-Dates Babies at the Age of Four Years: Health, Handicap and Developmental Status.** *Early Human Development* 8(3/4): 243-258, October 1983.

In 244 average-for-date (AFD) children, those born to nonsmokers had higher scores in all sectors, with significant differences in gross motor and visuomotor function, language, and comprehension. No differences were found between the scores for 221 children born to smokers and nonsmokers who were small-for-date (SFD). All children were seen regularly from birth to 4 years of age. In the AFD group, differences were found in total scores according to social class, sex, and gestational age as well as smoking. In the SFD group, social class and method of delivery were the only factors that had a significant effect. The contributions made to developmental scores by gestational age and smoking were significantly different in the two groups, and operated in opposite directions.

When all children were considered and birth weight group included as an additional variable, no significant differences remained between the SFD and AFD groups for any sector of development.

84–1173. Peters, T. J.; Golding, J.; Butler, N. R.; Fryer, J. G.; Lawrence, C. J.; Chamberlain, G.V.P. ***Plus ca Change*: Predictors of Birthweight in Two National Studies.** *British Journal of Obstetrics and Gynaecology* 90(11): 1040-1045, November 1983.

In the British Perinatal Mortality Survey of 16,989 singleton births during 1 week of March 1958, analysis of covariance showed that major factors associated with birth weight of the infant were maternal height, history of smoking in pregnancy, parity, and history of pre-eclampsia during pregnancy. The same analysis (by the British Births Survey) on data for 16,792 singletons born 12 years later during 1 week of April 1970 showed that, during the 12 years, the proportion of women who smoked during pregnancy increased by a third. The interactions between parity and smoking, and pre-eclampsia and smoking, were significant in the 1970 study. Also, there was some indication that the birth weight difference between infants of smokers and nonsmokers had increased slightly. It is noted that despite major changes in obstetric practice and in the maternal population, the same factors were highly significant and there was little change in the magnitude of the associations. (Auth. Abs. Mod.)

84–1174. Trundle, J. I.; Skellern, G. G. **Gas Chromatographic Determination of Nicotine in Human Breast Milk.** *Journal of Clinical and Hospital Pharmacy* 8(3): 289-293, September 1983.

Nicotine was detected in the milk of all smoking mothers, with the exception of one mother who had not smoked on the day of sample collection. Human breast milk concentrations of nicotine were measured 1 to 10 days postpartum in hospitalized smoking and nonsmoking mothers (study 1) and in hospitalized and at-home smoking and nonsmoking mothers (study 2). Variables included time interval between smoking and sample collection, number of cigarettes already smoked on the day of sample collection, and average number of cigarettes smoked/day. Samples were analyzed by gas chromatography. Nicotine concentration ranged from 0 to 102 ng/ml, with no significant differences between the two study groups. Nicotine was also detected in samples from approximately 80 percent of nonsmokers who were hospitalized when samples were collected, at concentrations ranging from 0 to 25 ng/ml. In samples taken from nonsmokers in their own homes, nicotine was detected in only 12 percent (concentrations <8 ng/ml). It is concluded that lactating mothers should be advised to smoke as few cigarettes as possible to minimize the amount of nicotine ingested by infants via breast milk. (Auth. Abs. Mod.)

84–1175. Werner, E. J.; Stockman, J. A., III. **Red Cell Disturbances in the Feto-Maternal Unit.** *Seminars in Perinatology* 7(3): 139-158, July 1983.

Both maternal and fetal carboxyhemoglobin (COHb) levels at delivery are directly related to maternal cigarette consumption. Fetal hemoglobin appears to have a higher affinity for carbon monoxide; at comparable tensions, the fetal COHb level is 1.8 times that of the maternal level. Thus a 9 percent fetal COHb level (realistic for the fetus of a moderate smoker) is about equivalent to a 41 percent reduction in fetal oxygenation. The lower maternal pO_2 and nicotine-related reduced placental blood flow also impair fetal oxygenation. The fetus adapts to maternal smoking via an elevation of hematocrit. Pregnancy-associated red blood cell changes are also discussed with respect to nutrition, maternal anemia, red blood cell enzyme disorders, hypoplastic and immune hemolytic anemia, and the consequences of maternal hemoglobinopathies.

84–1176. Young, K. R. **Effect on Birth Weight of Smoking in Pregnancy.** *Journal of the Royal Army Medical Corps* 129(2): 101-103, June 1983.

Maternal cigarette consumption during pregnancy and infant birth weight were assessed in 427 consecutive births at a British military hospital. Of the 197 (46.1 percent) of mothers who smoked, 28.9 percent smoked <10 cigarettes/day, 50.2 percent between 11 and 20/day, and 20.8 percent >20/day. Only 39.3 percent of the 177 primagravidas smoked, but 52.3 percent of the 174 secundigravidas were smokers. Babies born to mothers who smoked, in each consumption group, were significantly lighter than those born to nonsmokers, regardless of birth order. There was no significant difference between the mean birth weight of babies in birth order one and birth order two, regardless of the mother's smoking status, but there was a significant difference within each birth order cohort. Of 24 low birth weight babies (<2.5 kg), 19 had mothers who smoked. Fourteen of these were preterm, and all but three were born to smokers. It is concluded that those involved in obstetric care, particularly in outposts that lack access to neonatal intensive care facilities, should spread the stop smoking message as forcefully as possible.

See also **84–1247**

OTHER DISEASES AND CONDITIONS

84–1177. Ageel, A. M.; Parmar, N. S.; Tariq, M. **The Effect of Nicotine Pretreatment on the Gastric Mucosal Damage Induced by Aspirin and Reserpine in Rats.** *Life Sciences* 34(8): 751-756, February 20, 1984.

Results show that nicotine administration (5 μ/ml in drinking water ad libitum) for 10 days significantly augments the aspirin- and reserpine-induced gastric mucosal damage in rats. Thirty-six Wistar albino rats were randomly assigned to (1) control (normal); (2) nicotine control; (3) aspirin alone; (4) nicotine and aspirin; (5) reserpine alone; and (6) nicotine and reserpine. Aspirin and reserpine administration resulted in the production of gastric mucosal damage mainly in the glandular segment of the stomach in all animals. Nicotine-pretreated animals in both groups developed significantly more severe damage compared with controls. It is suggested that the previously shown association between cigarette smoking and peptic ulcer development may be due to the effect of nicotine in association with other agents. Nicotine may possibly sensitize the mucosal cells to the ulcerogenic effects of aspirin. (Auth. Abs. Mod.)

84–1178. Araki, S.; Murata, K.; Ushio, K.; Sakai, R. **Dose-Response Relationship Between Tobacco Consumption and Melanin Pigmentation in the Attached Gingiva.** *Archives of Environmental Health* 38(6): 375-378, November-December 1983.

The dose—response relationship between tobacco consumption and melanin pigmentation in the attached gingiva was examined on two occasions (4 years apart) in a group of Japanese lead workers with blood lead concentrations below 2.0 μmol/kg. In the two examinations, 12 and 18 percent of the total group of workers, and 17 and 24 percent of the smokers had melanin pigmentation. The prevalence of pigmentation was significantly higher in smokers who inhaled more than 10 cigarettes/day (17 and 25 percent) than in nonsmokers (0 and 2 percent), and tended to increase significantly with tobacco consumption. Lead absorption, however, was not associated with melanin pigmentation. (Auth. Abs.)

84–1179. Arendorf, T. M.; Walker, D. M.; Kingdom, R. J.; Roll, J.R.S.; Newcombe, R. G. **Tobacco Smoking and Denture Wearing in Oral Candidal Leukoplakia.** *British Dental Journal* 155(10): 340-343, November 19, 1983.

Smoking >20 cigarettes/day and continuously wearing dentures seem to be important local factors in the etiology of oral candidal leukoplakia (chronic hyperplastic candidosis or CL) in adults. Clinical and histological features of 40 CL patients who smoked (82.5 percent heavy smokers) were compared with 40 normal subjects (18 smokers, 10 classifed as heavy smokers) and oral lichen planus (LP) patients (19 smokers), all matched for age and sex. Of CL patients, 36 smoked and wore dentures, compared with 9 LP patients and 14 normal subjects. While all 36 CL patients wore dentures continuously (day and night), only 15 normal subjects did so (data for LP patients were unavailable). It is suggested that tobacco smoking and denture friction alter the oral mucosal surface, facilitating candidal colonization, and that removal of CL lesions without addressing the local factors (smoking and continuous denture wearing) invites recurrence.

84–1180. Boysen, M.; Solberg, L. A.; Torjussen, W.; Poppe, S.; Hogetveit, A. C. **Histological Changes, Rhinoscopical Findings and Nickel Concentration in Plasma and Urine in Retired Nickel Workers.** *Acta Oto-Laryngologica* 97(1/2): 105-115, January-February 1984.

Histological examination of nasal biopsy specimens, rhinoscopic evaluation, and nickel analysis in plasma and urine of 59 retired nickel workers (21 smokers and snuff-dippers) showed that average histological score (based on a numerical scale from 0=pseudostratified columnar epithelum, to 7=carcinoma) and percentage of dysplasia (n=13) were 4.0 and 39 in smokers, compared with 2.8 and 13 in nonsmokers, respectively. Mean histological score and tobacco consumption were significantly correlated. In eight never smokers, the mean histological score was 2.4, compared with 3.3 in current and ex-smokers combined. Of four patients with nasal carcinoma, three were current smokers and the fourth was a heavy smoker who had quit. Multiple regression analysis indicated that tobacco smoking explained 14 percent of the histological score. Significantly lower mean histological scores were correlated with short employment and short retirement time.

84–1181. Fedullo, A. J.; Swinburne, A. J. **Distribution of Bacteria Adherent to the Buccal Epithelial Cells of Cigarette Smokers. (Letter).** *Journal of Infectious Diseases* 149(1): 122-123, January 1984.

An exponential distribution pattern of bacteria among the buccal epithelial cells (BEC) was observed in a group of 10 asymptomatic cigarette smokers. The adherence of indigenous gram-positive cocci was measured by counting under a microscope the number of bacteria adherent to 50 BECs before and after in vitro incubation with *S. pneumoniae* type 25. The mean in vivo adherence values ranged from 1.8 to 21.5 bacteria/cell (8.9±6.2). The mean adherence after in vitro incubation ranged from 2.9 to 50.4 bacteria/cell (24.7±14.4). Correlation coefficients for the regression of $\log_e$ number of cells on the percentage mean adherence (up to 500 percent) were -0.93 for the in vivo adherence and -0.94 for the adherence pattern after incubation, indicating a good quality of fit to an exponential curve ($p<0.01$).

84–1182. Goldstein, I.; Feldman, M. I.; Deckers, P. J.; Babayan, R. K.; Krane, R. J. **Radiation-Associated Impotence. A Clinical Study of Its Mechanism.** *Journal of the American Medical Association* 251(7): 903-910, February 17, 1984.

Smoking was significantly ($p=0.016$) associated with radiation-induced impotence (RAI) in 23 patients (mean age, 65 years) who received radiation therapy for prostate cancer. Of the 15 patients who experienced changes in erectile potency following radiotherapy, 14 (93 percent) were smokers. In contrast, only one (25 percent) of the four patients with unchanged erectile capacity was a cigarette smoker. Among the 15 with erectile potency changes, neurological, endocrine, and vascular testing revealed abnormalities in 0, 5, and 15 patients, respectively. In two, arteriography revealed bilateral occlusive disease in the distal internal pudendal and penile arteries overlying the pelvic radiation field. Thus vasculogenic impotence is the most consistent organic erectile abnormality in RAI. (Auth. Abs. Mod.)

84–1183. Hetzel, D. J.; Korman, M. G.; Hansky, J.; Shearman, D. J.; Eaves, E. R.; Schmidt, G. T.; Hecker, R.; Fitch, R. J. **The Influence of Smoking on the Healing of Duodenal Ulcer Treated With Oxmetidine or Cimetidine.** *Australian and New Zealand Journal of Medicine* 13(6): 587-590, December 1983.

Results from a double blind study of 83 duodenal ulcer patients randomly allocated to oxmetidine or cimetidine treatment indicated that ulcer healing was significantly less frequent in smokers (69 percent) than nonsmokers (89 percent) after 4 weeks. Smokers and nonsmokers were similar clinically and did not differ in compliance with medication. Smokers were not advised to change their smoking habits. Both H_2-receptor antagonists were equally effective, and ulcers were healed in 76 percent of patients after 4 weeks of treatment. Smokers with healed ulcers consumed fewer cigarettes/day (15.8 ± 1.4) than those whose ulcers did not heal (22.2 ± 2.7). When smokers were grouped according to daily cigarette consumption, a direct relationship was found between increasing cigarette consumption and decreasing frequency of ulcer healing. No reduction in ulcer healing was apparent in patients who smoked nine or less cigarettes/day. (Auth. Abs. Mod.)

84–1184. Holdstock, G.; Savage, D.; Harman, M.; Wright, R. **Should Patients With Inflammatory Bowel Disease Smoke?** *British Medical Journal* 288(6414): 362, February 4, 1984.

Smokers with Crohn's colitis had more relapses and more severe pain than nonsmokers, while smokers with small bowel Crohn's disease had more frequent bowel movements, more admissions to hospital, more operations, and higher white cell counts. Interviews were conducted with 102 outpatients with ulcerative colitis, 96 with Crohn's colitis, and 54 with Crohn's disease of the small bowel and case notes reviewed. Of the ulcerative colitis patients, 8 percent were smokers and 32 percent were ex-smokers, compared with 25 percent smokers and 30 percent ex-smokers in the Crohn's colitis group, and 52 percent smokers and 13 percent ex-smokers among those with small bowel Crohn's disease. Of the ulcerative colitis patients, 9 of the 13 who reported continuous symptoms were smokers, compared with only 11 of 49 patients who suffered less than one relapse/year. It is suggested that smoking influences the pathological appearances of the disease, leading to Crohn's disease rather than ulcerative colitis.

84–1185. Kellow, J. E.; Barr, G. D.; Cowen, A. E.; Ward, M.; Wood, L.; Piper, D. W. **Comparison of Ranitidine and Cimetidine in the Treatment of Chronic Gastric Ulcer.** *Digestion* 27(2): 105-110, June 1983.

Smoking habits did not influence healing rates in a comparison of the effectiveness of ranitidine and cimetidine in the treatment of chronic gastric ulcer in 44 outpatients (28 smokers, 63.6 percent). Patients were randomly allocated to receive either ranitidine, 150 mg twice a day or cimetidine, 200 mg three times a day and 400 mg at night for 4 weeks. If the ulcer had not healed, treatment was continued for 4 weeks, when patient assessment and endoscopy were repeated. By 4 weeks, 13 of 22 patients taking ranitidine and 14 of 22 patients taking cimetidine had healed. At 8 weeks, 91 percent on ranitidine and 91 percent on cimetidine had healed. Healing rates were similar in the smoking and in the nonsmoking patients, and in those who consumed >40 g alcohol/day and those who did not. Symptomatic improvement was similar with both drugs, and no major side effects occurred with either medication. (Auth. Abs. Mod.)

84–1186. Kikendall, J. W.; Evaul, J.; Johnson, L. F. **Effect of Cigarette Smoking on Gastrointestinal Physiology and Non-Neoplastic Digestive Disease.** *Journal of Clinical Gastroenterology* 6(1): 65-79, February 1984.

In a literature review of the relationship of smoking and nonneoplastic diseases of the gastrointestinal tract, it is shown that smoking enhances all the known mechanisms active in the pathogenesis of reflux esophagitis. Cessation of smoking might reduce reflux symptoms in otherwise healthy patients and those with complications. Smoking is also linked to duodenal ulcer, although the relationship with gastric ulcer is still only suggestive. Evidence indicates that smoking significantly alters the clinical pharmacologic effect of certain drugs such as pentazocine and theophylline on hepatic function, and it is possible that hypoxia associated with smoking could adversely affect the clinical course of alcoholic liver disease. It is hoped that the information provided will help physicians in counseling patients. The need for additional research is discussed, but current evidence warrants advising patients that smoking may be partly responsible for gastrointestinal symptoms and ultimate complications.

84–1187. Lamid, S.; El Ghatit, A. Z. **Smoking, Spasticity and Pressure Sores in Spinal Cord Injured Patients.** *American Journal of Physical Medicine* 62(6): 300-306, December 1983.

Cigarette smoking was associated with a higher incidence and more extensive pressure sores in the 17 paraplegics and 21 quadraplegics surveyed. The male patients, aged 21 to 68, were evaluated for pack-years of smoking, muscle spasticity, body weight, type of wheelchair cushion, and various life activities, in addition to type and frequency of pressure ulceration. Smoking was correlated with pressure sores ($r=0.47$), but not to spasticity. Patients with higher pack-years of smoking had higher scores of pressure sores, a significant dose—response effect.

84–1188. Littlewood, J. T.; Glover, V.; Sandler, M.; Petty, R.; Peatfield, R.; Rose, F. C. **Migraine and Cluster Headache: Links Between Platelet Monoamine Oxidase Activity, Smoking and Personality.** *Headache* 24(1): 30-34, January 1984.

A possible genetic connection between predisposition to smoke, low platelet monoamine oxidase (MAO) activity, and headache was investigated. The incidence of smoking among 35 male cluster headache patients was significantly greater (95 percent) than in a matched group of 53 male migrainous patients (26 percent). Both groups had significantly lower MAO activity than controls, where MAO was expressed in nmoles tyramine oxidized/mg protein/30 min. This finding may be connected genetically with certain personality characteristics, including the predisposition to smoke. It is possible that patients with migraine headaches smoked less than cluster headache patients because the process of smoking has a greater tendency to provoke headache attacks in this group. (Auth. Abs. Mod.)

84–1189. Lombrail, P.; Passa, Ph.; Thibult, N.; Eschwege, E.; Canivet, J. **Prevalence du Tabagisme Chez les Diabetiques et Influence du Tabac sur la Retinopathie. [Prevalence of Smokers Among Diabetics and Influence of Tobacco on Diabetic Retinopathy.]** *Presse Medicale* 12(42): 2677-2679, November 19, 1983, French.

Among 647 diabetics regularly followed who attended the outpatient clinic of a hospital Diabetology Department between December 1, 1979, and June 30, 1980, 35 percent were habitual smokers, 20 percent ex-smokers, and 45 percent nonsmokers. Insulin-treated diabetics did not smoke more than the noninsulin-treated diabetics. Only half of the smokers declared that they had been advised by a doctor to stop smoking. It is concluded that an effect of tobacco on the prevalence or severity of diabetic retinopathy was not demonstrated. (Auth. Abs. Mod.)

84–1190. Mangan, G. L.; Golding, J. F. **The Effects of Smoking on Memory Consolidation.** *Journal of Psychology* 115(First half): 65-77, September 1983.

The effects on memory of smoking deprivation and of smoking a single cigarette with a 0.8 mg (low), 1.3 mg (middle), or 2 mg (high) level of nicotine, immediately postacquisition of a paired-associate learning (PAL) task, were investigated in 54 male smokers and 15 male nonsmokers. Subjects were retested for retention of the memorized material at intervals of 30 minutes, 1 day, 1 week, and 1 month. Nonsmokers showed superior recall compared with all smokers at 30-minute retest and with some of the smoking groups on later retests. At 1-month retest, the low and middle nicotine smokers outperformed high nicotine smokers and also achieved superior recall compared with nonsmokers. Findings are discussed in terms of smoker versus nonsmoker differences, the effects of nicotine on memory consolidation, and the PAL response measure adopted. (Auth. Abs. Mod.)

84–1191. Massarrat, S.; Paidlik, A.; Pittner, P.; Schmitz-Moormann, P.; Wurbs, M. **The Role of Certain Habits and Various Diseases in the Occurrence of Gastritis.** *Hepato-Gastroenterology* 30(6): 249-253, December 1983.

In contrast to earlier reports, no increase in gastritis was found in smokers ($n=38$, consumption >20 cigarettes/day) compared with nonsmokers. Mean age-adjusted gastritis scores ranged from 1.6 to 2.5 in the antrum and fundus of smokers and from 1.7 to 2.3 in nonsmokers. Four biopsy specimens of antral and body mucosa were taken from the greater and lesser curvature of the stomach in 557 Germans and 46 Turks with no gastric or duodenal lesions. The age-adjusted gastritis score in each area was related to various habits and clinical diagnoses. Unlike the Germans, the Turks had advanced antral gastritis at an early age that did not progress with age. Patients of low social class were more prone to antral gastritis than academics and civil servants. Hypertension was associated with a lower incidence of antral gastritis. Patients with a history of cholecystectomy were more likely to have advanced gastritis in antral and body mucosa than those with gallstones but intact gallbladders. The incidence of fundic gastritis was higher in patients with degenerative joint disease than in controls. It is concluded that nicotine, alcohol, drug abuse, and various diseases do not play an important role in the frequency of occurrence of gastritis. (Auth. Abs. Mod.)

84–1192. Matsuo, K.; Kamimura, M.; Hirano, M. **Polypoid Vocal Folds. A 10-Year Review of 191 Patients.** *Auris, Nasus, Larynx* 10(Supplement): S37-S45, 1983.

A retrospective review of the clinical records of 191 patients with polypoid vocal folds, diagnosed over a 10-year period, found that smoking and aging are important causative factors. Of the 164 patients who reported smoking habits, only 13 were nonsmokers, and 105 smoked ≥ 20 cigarettes/day. In addition, results showed that (1) there was no marked difference in incidence between males and females; (2) incidence was high in those over age 40; (3) bilateral lesions were much more frequent than unilateral lesions; (4) long-lasting hoarseness was the commonest symptom; (5) vocal abuse, alcohol drinking, and air pollution did not prove to be etiologic factors; and (6) no significant differences in subjective voice improvement were found between the stripping and the sucking technique. (Auth. Abs. Mod.)

84–1193. Nagata, K.; Kurita, S.; Yasumoto, S.; Maeda, T.; Kawasaki, H.; Hirano, M. **Vocal Fold Polyps and Nodules. A 10-Year Review of 1,156 Patients.** *Auris, Nasus, Larynx* 10(Supplement): S27-S35, 1983.

Clinical record data on 784 patients with vocal fold polyps and 372 with vocal fold nodules showed no positive relationship between smoking and development of polyps or nodules. Only 5 percent of patients with nodules were smokers, and only 19 percent of the patients with polyps smoked more than 20 cigarettes/day. Polyps were frequent in middle-aged groups in both sexes, while nodules were common in boys and middle-aged women. Vocal abuse was the most important etiologic factor. The major treatments for vocal fold polyps and nodules are surgical removal and voice therapy. (Auth. Abs. Mod.)

84–1194. Rack, J.; Sonnenberg, A. **The Influence of Smoking and Intravenous Nicotine on Gastric Mucus.** *Hepato-Gastroenterology* 30(6): 258-260, December 1983.

Results showed that neither chronic nor acute consumption of nicotine affects gastric turnover or adherence of mucus to the mucosa. To examine whether smoking changes gastric mucus secretion, the concentration of N-acetylneuraminic acid (NANA) was measured in the gastric juice of 10 nonsmokers and 10 smokers. Gastric juice was aspirated during intravenous infusion of 0.67 µg/kg/hr pentagastrin given alone or together with 5 µg/kg/hr nicotine. Instead of intravenous nicotine, the 10 smokers smoked five cigarettes over a period of 2 hours. In the fasting gastric juice and during infusion of pentagastrin, the gastric concentration and output of bound NANA were similar in smokers and nonsmokers. Acute nicotine administered intravenously or by smoking left gastric output of bound NANA unaffected. (Auth. Abs. Mod.)

84–1195. Repace, J. L.; Lowrey, A. H. **Modeling Exposure of Nonsmokers to Ambient Tobacco Smoke.** Prepared for presentation at the 76th Annual Meeting of the Air Pollution Control Association, Atlanta, Georgia, June 19-24, 1983, 22 pp.

A model for determining the exposure of nonsmokers to indoor pollution from tobacco smoke is presented. The range of exposure has been estimated as up to 14 mg of tobacco tar/day. Based on weighted averages of exposure, it is now estimated that the average nonsmoker of working age receives an average of 1.4 mg tar/day. Individuals exposed to ambient smoke both at home and at work (approximately 39 percent of adult nonsmoking workers) receive an estimated daily average exposure of 2.4 mg/day. Those exposed to ambient smoke only at home (approximately 23 percent) receive an estimated 0.5 mg/day, while those exposed only at work (approximately 24 percent) have an average exposure of 1.8 mg/day. Thus, the ratio of workplace dosage to home exposure is nearly 4 to 1, indicating that the workplace is a more important source of the particulate phase of tobacco than the home environment.

84–1196. Russell, M.A.H.; Lebowitz, M. D. **Environmental Tobacco Smoke. 4.2. Effects on Health.** *European Journal of Respiratory Diseases* 65(Supplement 133): 140-142, 1984.

The consensus of a workshop group on the health effects of environmental tobacco smoke (ETS) are reported. It was agreed that annoyance was among the acute effects of exposure. Specific effects include eye and nose irritation. Published results on adult asthmatics are considered contradictory and inadequate, requiring further research. Other sensitive groups should be examined also, particularly children, since ETS exposure results are inconsistent and effects may be acute or chronic, leading to problems as adults. Data on other chronic effects, such as allergic sensitization, development of lung cancer, chronic lung disease, coronary artery disease, and pregnancy outcome effects are also felt to be inconclusive, on the basis of present studies. Various recommendations for further research are included.

84–1197. Rylander, R. **Environmental Tobacco Smoke. 5. Workshop Perspectives.** *European Journal of Respiratory Diseases* 65(Supplement 133): 143-145, 1984.

A workshop on environmental tobacco smoke (ETS) concluded that eye irritation and annoyance are the most prevalent effects resulting from exposure. Data suggested, although not conclusively, that other effects such as respiratory disease and lung cancer could occur in certain populations. However, a means of determining ETS exposure must be provided before health effects can be further evaluated. Under extreme conditions, daily ETS exposure could reach the equivalent of a few cigarettes, but a more realistic dose would probably be about 0.1 cigarettes. The unimportance of carbon monoxide from a health point of view is further confirmed. Information concerning the extent of irritation reactions in the general population under everyday conditions is still lacking, and it is thus premature to suggest maximal workplace exposure levels.

84–1198. Rylander, R.; Peterson, Y.; Snella, M.-C. (Editors). **ETS — Environmental Tobacco Smoke. Report From a Workshop on Effects and Exposure Levels.** *European Journal of Respiratory Diseases* 65(Supplement 133): 1-152, 1984.

The present workshop was held to evaluate and form conclusions concerning the data published since the 1974 workshop and to suggest areas of future research. Levels of exposure to environmental tobacco smoke (ETS) are explored with respect to measurement issues, chemical analysis of smoke constituents, the effect of regulating smoking in office buildings, and carbon monoxide as an index of ETS exposure. The problem of human exposure measurement is also discussed, including the validity of questionnaire data, and the half-lives of selected tobacco smoke exposure markers. In considering the effects of ETS exposure in humans, data on lung function, respiratory symptoms in children, lung cancer, acute effects of exposure in asthmatics and nonasthmatics, and mortality in Scotland are reviewed. See also 84–1000, 84–1002, 84–1008, 84–1017, 84–1024, 84–1029, 84–1048, 84–1079, 84–1089, 84–1109, 84–1120, 84–1196, 84–1197, 84–1202, 84–1226, 84–1315.

84–1199. Seeman, E.; Melton, L. J., III; O'Fallon, W. M.; Riggs, B. L. **Risk Factors for Spinal Osteoporosis in Men.** *American Journal of Medicine* 75: 977-983, December 1983.

The relative risk (RR) for osteoporosis, estimated by the odds ratio, is 2.3 among cigarette smokers, 2.4 for those who drink alcoholic beverages (greater still for those who both drink and smoke), and 5.5 among patients with a disease known to affect calcium or bone metabolism. Data were obtained from a 4-year study of vertebral fractures due to osteoporosis in 105 consecutive male patients. An equal number of men with Paget's disease, matched by age and concurrently attending the same subspecialty clinic, served as controls. Tobacco consumption expressed in pack-years was greater in the case group (79 percent) than in controls (63 percent). The risk associated with smoking increased by a factor of 1.009 for each pack-year of exposure, but this difference did not achieve statistical significance. As assessed by a multiple logistic model, the risk associated with smoking and drinking increased with age, but obesity was a protective factor. Effects of the four major risk factors were largely independent of one another and were cumulative. Thus, spinal osteoporosis in men is frequently associated with recognizable risk factors, some of which are potentially remediable. (Auth. Abs. Mod.)

84–1200. Telmer, S.; Christiansen, J. S.; Andersen, A. R.; Nerup, J.; Deckert, T. **Smoking Habits and Prevalence of Clinical Diabetic Microangiopathy in Insulin-Dependent Diabetics.** *Acta Medica Scandinavica* 215(1): 63-68, 1984.

Cigarette smoking was found to be a risk factor for the development of nephropathy in 688 patients with insulin-dependent diabetes mellitus (onset before age 30; duration of disease, 12 to 40 years). Patients answered a World Health Organization questionnaire on cigarette smoking, and smoking habits were compared with the frequency of proliferative retinopathy and/or diabetic nephropathy (persistent proteinuria). The prevalence of diabetic nephropathy was significantly higher among heavy smokers (>10 cigarettes/day for more than 1 year) than among light smokers (19.2 percent versus 12.1 percent). An increasing frequency of nephropathy was found with increasing cigarette consumption, but no association between cigarette smoking and proliferative retinopathy could be demonstrated. Significantly more females than males were never smokers, while 19.2 percent of the men and 3.4 percent of the women were pipe and/or cigar smokers. Among current smokers, significantly more men than women smoked ≥20 cigarettes/day within all diabetes duration groups. (Auth. Abs. Mod.)

84–1201. Weber, A. **Luftungsmassnahmen zum Schutze der Passivraucher. [Ventilation Measures for the Protection of the Passive Smoker.]** *Haustechnik Bauphysik Umwelttechnik* 104(1): 37-42, January-February 1983, German.

Results of laboratory studies and field tests indicate that protection from passive smoking is necessary when carbon monoxide air concentrations rise above 2.0 ppm and recommended at levels above 1.5 ppm. Field tests attribute from 30 to 70 percent of the measured air pollutant concentrations in occupational environments and restaurants to tobacco smoke. Of persons questioned in the workplace, 40 percent felt bothered by cigarette smoke and 30 percent suffered from eye irritation, compared with 28 and 12 percent of restaurant patrons, respectively. Organizational measures to protect passive smokers, such as separate smoking/nonsmoking areas, partial or total smoking bans, and/or appropriate ventilation are recommended. Results of various studies suggest that inflammatory reactions and respiratory problems in children and patients suffering from allergies, asthma, and circulatory illnesses, as well as restricted respiratory function and increased lung cancer risk, may be related to chronic passive smoke exposure.

84–1202. Weber, A. **Environmental Tobacco Smoke. 3.4. Acute Effects of Environmental Tobacco Smoke.** *European Journal of Respiratory Diseases* 65(Supplement 133): 98-108, 1984.

Results of a laboratory study that exposed 33 healthy students to varying levels of sidestream smoke showed that eye irritation and mean eye blink rate increased in almost linear fashion with increasing smoke concentration. In a second experiment, eye irritation increased with duration of exposure despite a constant smoke concentration. Duration of exposure had almost no influence on degree of annoyance. The third experiment (33 subjects), to determine the components of tobacco smoke responsible for irritation, found that acrolein and formaldehyde had little effect on irritation levels. Gas phase compounds were largely responsible for reported annoyance, and the particulate phase was found to be the main cause of the irritating effects of sidestream smoke. In a field study of 44 workrooms, it was found that (1) approximately one-third of employees in workplaces where ETS was present labeled the air as "bad"; (2) 40 percent were disturbed by the smoke; (3) 25 percent reported eye irritation at work; and (4) nonsmokers reacted significantly more than smokers. Also those with hay fever showed higher irritation levels. Psychological factors (general job satisfaction or relationships with coworkers) may influence an individual's evaluation of irritation and annoyance. The problems of using nicotine and CO as indicators of ETS are discussed. It is suggested that a limit of between 1.5 and 2.0 ppm CO in workplace environments would protect healthy people against impairment of well-being.

84–1203. Wilson, J. **Cyanide in Human Disease: A Review of Clinical and Laboratory Evidence.** *Fundamental and Applied Toxicology* 3(5): 397-399, September-October 1983.

For transmitters of Leber's hereditary optic atrophy, smoking carries the risk of blindness, and in severely affected patients, there is diffuse neurological disease. In such subjects, there appears to be a defect in the conversion of cyanide to thiocyanate because of deficient rhodanese activity. Experimental cyanide exposure in animals causes demyelination, which may have human parallels. It is possible that other hereditary optic atrophies (dominant and recessive) may also reflect inborn errors of cyanide metabolism. In retrobulbar neuritis and optic atrophy of vitamin B_{12} deficiency there may be a conditional abnormality of cyanide metabolism in smokers, also a possibility in so-called tobacco-alcohol amblyopia in which there are more complex nutritional deficiencies. Epidemiological evidence (differing sex ratios, excess of smokers) indicates that defective cyanide metabolism may contribute to the development of sub-acute combined degeneration of the cord in vitamin B_{12} deficiency. The known pathways of human cyanide metabolism are reviewed and evidence supporting the clinical data is presented. (Auth. Abs. Mod.)

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

84–1204. Abelin, T.; Mueller, R. **Trend der Rauchgewohnheiten in der Schweiz 1975—1981. [Trend of Smoking Habits in Switzerland 1975—1981.]** *Sozial- und Praventivmedizin* 28(3/4): 185-195, 1983, German.

Data from sample surveys (1975 and 1981) of German- and French-speaking adults in Switzerland show that, among men aged 15 to 74 years, there was a 12 percent decrease in the number of smokers (including smokers of pipes and cigars). Among women, the number of smokers stabilized at 28 to 29 percent. The remarkable overall decrease of more than 20 percent in the smoking rates among men aged 35 and over is noted. A higher proportion of men and women below the age of 35 are smokers in the French-speaking areas compared with the German-speaking parts. It is noted that the smoking rates in the Italian-speaking area lie between those of the other two linguistic regions. The proportion of moderate smokers (one to nine cigarettes/day) decreased, while the proportion of heavy smokers (≥20 cigarettes/day) increased, particularly among women. The Italian-speaking men had the highest percentage of heavy smokers. Among all smokers, about 40 percent claim to have tried to quit, and it is believed that about half would like to quit. Approximately 75 percent of all adults were in favor of creating nonsmoking areas in restaurants, support being strongest (especially among men) in the German-speaking areas. It is concluded that increased financial aid will be required to reach the younger age groups and women, through health education and other programs, to counter the effect of cigarette advertisements. In such programs, different approaches will be needed for each linguistic region.

84–1205. Billings, A. G.; Moos, R. H. **Social-Environmental Factors Among Light and Heavy Cigarette Smokers: A Controlled Comparison With Nonsmokers.** *Addictive Behaviors* 8(4): 381-391, 1983.

Data from a study of the role of psychosocial factors among a representative community sample of both smokers (52 light and 127 heavy) and nonsmokers (414) suggest that psychosocial influences may have differential relevance during the different phases of smoking behavior (initiation, maintenance, and cessation). Support for the importance of psychosocial influences on smoking is limited because the evidence is primarily based on intervention studies that deal with self-selected and thus unrepresentative samples of smokers. There have been few comparisons with nonsmokers on these factors, but in this study, the role of psychosocial factors among a representative community sample of smokers and nonsmokers was examined. Heavy smokers were distinguished from nonsmokers by only slightly higher levels of environmental stressors, less supportive social resources, and poorer psychological functioning. Light smokers did not differ from nonsmokers on these factors. Although stressors and resources were correlated with psychological functioning across all respondents, such relationships were not significantly stronger for either heavy or light smokers. (Auth. Abs. Mod.)

84–1206. Bobon, D. **Developpement et Entretien de la Pharmacodependance Nicotinique. [Development and Care of Pharmacological Nicotine Dependence.]** *Revue Medicale de Liege* 38(17): 647-651, September 1, 1983, French.

The mechanism of action of nicotine is complex. Rats given low doses have increased spontaneous motor activity and explorations and more rapid learning of labyrinths. These stimulation effects have been confirmed in human studies. Nicotine dependence is compared with pharmacological dependence, since underlying causes and withdrawal symptoms are similar to those associated with alcohol and drug abuse. It has been shown that people who abuse other substances are, almost without exception, also cigarette smokers. In one study, 13 percent of schoolchildren who smoked had also experimented with drugs at least once, compared with only 3 percent of the nonsmokers. Smoking initiation has been related to imitation of adults or friends, engaging in a forbidden activity, various personality traits, and socioeconomic status. It is suggested that smoking appears to be an inexpensive status symbol, and that people at higher economic or educational levels are more likely to abstain or quit. Cessation of smoking is associated with irritability and weight gain. The obsessive desire for a cigarette, which is strongest during the first 6 months, may persist for years.

84–1207. Bradley, N.C.A. **Sale of Cigarettes to Children in Exeter.** *Journal of the Royal College of General Practitioners* 33(254): 559-562, September 1983.

To investigate cigarette sales to children in Exeter, 37 local shops were visited and the salespeople were interviewed: 28 incorrectly understood the relevant law and 7 had no knowledge of it. Only seven people said they tried to keep within the law. Only 2 of the 37 shops displayed the Tobacco Advisory Council's explanatory notice sent to every cigarette retailer in the United Kingdom in 1981, although every shop sold cigarettes. It is proposed that (1) a notice explaining the law be displayed in all cigarette retailing shops; (2) the tobacconist's license (abolished in 1963) be reintroduced; and (3) local civic and health authorities combine to educate retailers, their staffs, and the public. (Auth. Abs.)

84-1208. Cohen, S.; Kamarck, T.; Mermelstein, R. **A Global Measure of Perceived Stress.** *Journal of Health and Social Behavior* 24(4): 385-396, December 1983.

Evidence is presented from three samples (two of college students and one of 64 participants in a community smoking cessation program), for the reliability and validity of the 14-item Perceived Stress Scale (PSS), designed to measure the perceived stressfulness of life situations. The PSS showed adequate reliability and, as predicted, was correlated with life-event scores, depressive and physical symptomatology, utilization of health services, social anxiety, and smoking reduction maintenance. The 27 males and 37 females participating in the smoking cessation program had been smoking for an average of 20.9 years. Following 6 weeks of treatment, 64 percent were abstinent. During pretreatment, subjects completed a life-event scale, a physical symptom checklist, and the PSS; the physical symptom checklist and the PSS were repeated following treatment. A four-item version of the PSS was used for telephone interviews at 1 month and 4 months posttreatment. Analyses indicate that changes in perceived stress as measured by the PSS are predictive of changes in smoking rate. In all comparisons, the PSS was a better predictor of the outcome in question than were life-event scores. (Auth. Abs. Mod.)

84–1209. Flay, B. R.; D'Avernas, J. R.; Best, J. A.; Kersell, M. W.; Ryan, K. B. **Cigarette Smoking: Why Young People Do It and Ways of Preventing It.** In: McGrath, P. J.; Firestone, P. (Editors). *Pediatric and Adolescent Behavioral Medicine*, Springer Series on Behavior Therapy and Behavioral Medicine, Volume 10, New York, Springer Publishing Company, 1983, pp. 132-183.

Most recent research has indicated that a wide range of smoking cessation techniques are ineffective and that, in recent years, smoking in younger children has increased as the age of onset has decreased. This portends increasing health problems in future generations and suggests the need for programs to prevent or delay the onset of regular smoking in adolescents. Literature on the process of becoming a smoker is reviewed and used to derive a new developmental causal model that reflects the influence of family, peer pressure, self-image, and psychological attitudes. School-based attempts to prevent adolescent smoking are also examined, with emphasis on two major types of intervention: traditional educational programs that rely on disseminating information; and newer programs, based on social psychological theories that attempt to provide adolescents with decisionmaking and social skills to help resist the pressures to smoke. Components of one prevention program designed to be consistent with the known correlates (and suspected causes) of smoking and based on the new developmental model of attitude and behavior change are described, along with recent results. These observations on the antecedents of smoking and the process of becoming a smoker are integrated to derive implications for future program development and research.

84–1210. Gunn, R. C. **Does Living With Smokers Make Quitting Cigarettes More Difficult?** *Addictive Behaviors* 8(4): 429-432, 1983.

Results from clinical smoking cessation programs indicate that giving up a smoking addiction requires inner motivation from the smoker, with external cues for smoking and support for cessation having minimal influence. The assumptions that living with smokers and having housemates with little interest in one's stopping smoking would make quitting cigarettes difficult were tested by comparing successful quitters with nonquitters for housemates' smoking habits and attitudes. Subjects were 147 participants of several American Cancer Society Stop Smoking Clinics. Chances of quitting were not enhanced by living either with nonsmokers or with people believed to have a high interest in one's quitting, regardless of the closeness of the relationship. (Auth. Abs. Mod.)

84–1211. Henningfield, J. E.; Chait, L. D.; Griffiths, R. R. **Effects of Ethanol on Cigarette Smoking by Volunteers Without Histories of Alcoholism.** *Psychopharmacology* 82(1/2): 1-5, 1984.

The effects of alcohol on cigarette smoking were assessed in volunteer research subjects who had histories of light to moderate social drinking. Five subjects participated individually in daily 90-min sessions conducted in rooms equipped to automatically monitor cigarette smoking behavior. Each subject was tested at four dose levels of alcohol and placebo, which were given orally on a double-blind basis, 30 min prior to sessions. Dose order was according to a random block sequence in which each dose was given in each of five blocks of five sessions. Data from five alcoholic subjects who were similarly tested at only one alcohol dose level were used for comparison. For the nonalcoholic group, alcohol doses that produced reliable changes in group scores on various psychometric instruments produced no significant change in smoking behavior. There were differences among the nonalcoholic subjects, however, in that smoking was significantly decreased by alcohol in two subjects, increased in two subjects, and unchanged in the fifth subject. For the alcoholic group, alcohol produced reliable changes in psychometric measures and significant increases in cigarette smoking. Within- and between-group analyses of results suggest that the effect of alcohol on cigarette smoking may be related to prior history of alcoholic beverage consumption. (Auth. Abs.)

84–1212. Henningfield, J. E.; Miyasato, K.; Jasinski, D. R. **Cigarette Smokers Self-Administer Intravenous Nicotine.** *Pharmacology Biochemistry and Behavior* 19(5): 887-890, November 1983.

Male volunteers (n=6, mean age 36, all smokers) were equipped with i.v. catheters for seven 3-hour sessions and allowed to inject themselves with nicotine (1.5 mg/injection) as desired. Structured questionnaires were administered to assess alterations in subjective states accompanying injections. Nicotine injections were taken in orderly patterns that were related to unit dose. The four subjects with histories of drug dependence (including cocaine abuse) identified the injected nicotine as cocaine. Both euphoric and dysphoric effects were reported. Saline was also self-administered, but differed from nicotine by (1) not producing subjective effects as measured by the psychometric instruments, (2) occurring in highly variable patterns, and (3) decreasing in frequency as the session progressed. It is concluded that patterns of nicotine self-administration share commonalities with i.v. administration of other drugs of abuse, and that nicotine functions as an integral part of the behavioral process of compulsive tobacco use.

84–1213. Johnson, M.R.D.; Murray, M.; Bewley, B. R.; Clyde, D. C.; Banks, M. H.; Swan, A. V. **Categorie Sociale, Parents, Enfants et Tabagisme. [Social Class, Parents, Children and Smoking.]** *Bulletin de l'Union Internationale Contre la Tuberculose* 57(3/4): 267-271, September-December 1982, French.

Data indicate that smoking among children is significantly related to parents' smoking habits and social class. Questionnaires were answered by 6,330 students, aged 11 and 12, of the Derbyshire, United Kingdom, secondary schools and by 5,560 of their parents. Analysis of the parents' answers revealed that (1) significantly more parents of the manual working class (compared with the white-collar class) were current or ex-smokers; (2) of white-collar class fathers and mothers, 34 percent and 25 percent have been cigarette smokers vs. 56 and 42 percent, respectively, of the manual working class; and (3) significantly more white-collar class than manual working class parents have stopped all kinds of smoking. The incidence of regular or occasional smoking among the children tended to increase from white-collar to manual working class. The average cigarette consumption was 4.5 and 4.3/week for boys and girls, respectively, for all social classes. For children, risk of becoming a smoker was (1) about 1.25 times greater in manual working class families; (2) 1.25 times greater when parents were ex-smokers; and (3) 1.5 times greater when parents were current (compared with never) smokers. Children of a smoking mother and a nonsmoking father were the most exposed to the risk of smoking.

84–1214. Kandel, D. B. **Marijuana Users in Young Adulthood.** *Archives of General Psychiatry* 41(2): 200-209, February 1984.

Among 1,325 young adults (aged 24 to 25 years), 77 percent of the men who used marihuana ≥ 4 times/week reported lifetime consumption $\leq 1{,}000$ cigarettes compared with only 21 percent of those who had never used marihuana. Also, high-level marihuana users had smoked an average of 7.1 years, compared with 2.4 years among nonusers. The differences between marihuana users and nonusers increased with involvement, although no threshold appeared at any particular level. Marihuana users were characterized by higher use of other illicit substances in addition to tobacco, membership in networks of marihuana users, lower participation and greater instability in conventional roles of adulthood, history of psychiatric hospitalization and lower psychological well-being, and participation in deviant activities. Involvement with marihuana-using friends, use by spouse or partner, and use of other illicit drugs were important predictors of current marihuana involvement. It is noted that when the data were stratified for sex and living arrangement, lifetime cigarette use was still significantly correlated with marihuana use. (Auth. Abs. Mod.)

84–1215. Khavari, K. A.; Douglass, F. M. **Empirically-Derived Hierarchy of Use for Psychotropics: A Cost Benefit Index.** In: Eiseman, S.; Wingard, J. A.; Huba, G. J. (Editors). *Drug Abuse. Foundation for a Psychosocial Approach.* Farmingdale, New York, Baywood Publishing Company, 1984, pp. 173-178.

It is proposed that an individual's decision to use drugs is reached through a rational, complex process of cost—benefit analysis (psychological, physiological, social, and economic costs vs. perceived benefits such as euphoria, pleasure, and relaxation). Based on survey data from 1,121 adults from varied educational and occupational backgrounds, a cost—benefit index and a hierarchical order of use of tobacco, alcohol, marihuana, and 12 other drugs was devised. Survey data revealed that 99 percent of the population had tried alcohol, 77 percent had tried tobacco, and 62 percent had experimented with marihuana. Likewise, 94 percent continued to consume alcohol, 39 percent were regular smokers, and 6 percent reported daily marihuana use. The cost—benefit index is defined as a dispersion unit (DU), which reflects the difference between two adjacent levels of drug use in terms of derivation from the mean. Thus the DU for tobacco is 3.52, compared with 5.27 for marihuana, 7.11 for alcohol, 13.89 for cocaine, and 30.14 for methadone. A numerically small DU represents a low-cost high-benefit ratio, as determined by actual use.

84–1216. Kozlowski, L. T. **Perceiving the Risks of Low-Yield Ventilated-Filter Cigarettes: The Problem of Hole-Blocking.**

In: Covello, V. T.; Flamm, W. G.; Rodricks, J. V.; Tardiff, R. G. (Editors). *The Analysis of Actual Versus Perceived Risks*, Advances in Risk Analysis, Volume 1, New York, Plenum Press, 1983, pp. 175-182.

Some smokers of low-yield ventilated-filter cigarettes derive higher levels of tar, nicotine, and carbon monoxide than advertising or government smoking machine assays lead them to believe. A form of compensatory smoking involving blocking of the ventilation holes is a technique occurring with alarming frequency among smokers of cigarettes with <7 mg tar. In a sample of 39 observed smokers of ventilated-filter cigarettes, 44 percent blocked the ventilation holes with lips or fingers, thus defeating one of the major strategies employed by cigarette manufacturers to produce low-yield products. This compensatory smoking behavior was unintentional among 80 percent of the subjects. Tobacco consumers must therefore be educated on the role of air dilution holes in cigarettes and need to monitor the manner in which they smoke to prevent blockage of the holes.

84–1217. Ksir, C.; Benson, D. M. **Enhanced Behavioral Response to Nicotine in an Animal Model of Alzheimer's Disease.** *Psychopharmacology* 81(3): 272-273, October 1983.

Three groups of rats that received either injections of kainic acid (KA) or artificial cerebrospinal fluid in the ventral pallidum, or no operation, were tested in photocell activity cages following recovery from surgery. Locomotor activity was measured following injections of saline, nicotine (0.1, 0.2, 0.4 mg/kg) or *d*-amphetamine (0.5, 1.0 mg/kg). The KA rats showed an enhanced locomotor response after injections of nicotine compared with sham-operated or unoperated controls. In contrast, both lesioned and control rats showed increased activity after amphetamine; this effect was not influenced by the lesion. Since these lesions are known to produce neurochemical and cellular changes resembling those seen in human Alzheimer's disease, this increased response to nicotine might also be found in Alzheimer's patients and serve as the basis for a diagnostic test. (Auth. Abs.)

84–1218. Liddell, I.; Dewar, H. A. **Smoking Amongst Nurses in Newcastle in 1982.** *Public Health* 97(5): 246-256, September 1983.

In the autumn of 1981, 5,300 nurses employed by the Newcastle Area Health Authority were sent a letter and a booklet about smoking and advised to watch a television program about stopping smoking. A few months later, questionnaires administered to a sample of 285 (excluding males and auxiliaries) showed that, despite serious defects in distribution, the response had been favorable. It also showed that 30 percent were smokers and 16 percent ex-smokers, two-thirds of the smokers began before entering nursing, and nonsmokers exaggerated the importance of stress. Smokers resented and ignored exhortations to "set an example," but all respondents agreed that the considerations most likely to be effective in dissuasion are (1) the health risks, (2) the cost, and (3) the unpleasantness and social unacceptability of the habit. (Auth. Abs.)

84–1219. Mangan, G. L.; Golding, J. F. **Factors Underlying Smoking Recruitment and Maintenance Amongst Adolescents.** *Advances in Behaviour Research and Therapy* 4(4): 225-272, 1983.

A total of 2,302 comprehensive school students in Oxfordshire (12 to 17 years old) were surveyed to identify the sociocultural, psychobiological, and social learning variables that predict onset and persistence of smoking. Personality variables predictive of smoking were also explored. A 500-item questionnaire was first administered to 752 students. The remaining 1,550 subjects completed questionnaires developed from findings of the original survey that focused on specific age- or sex-related issues. Variables that discriminated between ever smokers and never smokers, and between smokers and ex-smokers (by stepwise multiple discriminant analysis), are summarized by age and sex. Among 12- to 13-year-olds, heavy smoking is symptomatic of pathology that penetrates deeply into the psychic structure. In ex-smokers of this age who do not report high levels of anxiety, heavy smoking appears to be one of a symptomatic cluster centering around rebelliousness to parents and authority. In 14- to 15-year-olds, some evidence of psychological disturbance was noted (particularly among boys who currently smoked), and items reflecting stimulation-seeking and aversive arousal control appeared. However, the 16- to 17-year-old smokers tested as relatively "normal," although showing high levels of alcohol consumption. Smoking typology and some current motivational models of smoking (including genetic, central arousal, psychobiological, social learning, and sociocultural theories) are reviewed.

84–1220. Manley, R. S.; Boland, F. J. **Side-Effects and Weight Gain Following a Smoking Cessation Program.** *Addictive Behaviors* 8(4): 375-380, 1983.

Abstainers reported increased appetite and overeating, while on all other side-effect variables, relapsers were found to be further from "optimal" than abstainers, lending support to the theory that cessation of smoking causes changes in eating behavior. Side effects and weight gain were assessed throughout and following a 4-week smoking cessation program with 94 subjects (39 males, 55 females; average age 37). Participants reported smoking an average of 23 cigarettes/day for 20 years, and had made previous attempts to quit. Subjects were randomly assigned to four different maintenance conditions: relapse prevention, cognitive urge control, behavioral urge control, and attention-placebo. Those in the cognitive urge condition were found to have gained significantly more weight compared with other conditions; however, this group also tended to have the highest abstinence rate and lowest percent baseline smoking at followup. Results are discussed within the context of incorporating side-effect control strategies as part of smoking cessation programs, noting that subjects should at least be told that they can probably expect a minor weight gain, which is unimportant compared with the dangers of smoking.

84–1221. Millar, W. J. **Prevalence of Smoking in the School Population of the Northwest Territories.** *Chronic Diseases in Canada* 4(3): 36-39, December 1983.

A November 1982 questionnaire survey of 7,305 students (70 percent response rate) in northwest Canada showed that overall, 28 percent of those aged 5 to 19 had smoked in the past 4 weeks and 17 percent smoked daily. While prevalence rates were higher among native youth, the frequency of smoking was greater among non-native youth: by age 8, 40 percent of Inuit youth had ever smoked, compared with 38 percent of Indian/Metis and 25 percent of non-native youth. Before age 10, boys were more likely to be regular smokers; however, after age 10, the smoking rate among girls was higher in each of the three ethnic groups. Girls were also more likely to smoke over 60 cigarettes/week. For students aged 15 to 19, the highest smoking rates were found among Inuit girls; 80 percent reported smoking in the past 4 weeks and 61 percent reported smoking daily, compared with 63 percent and 48 percent of other students, respectively. Among 10- to 14-year-olds, 27 percent were occasional and 13 percent were daily smokers. Also, 5 percent of those aged 5 to 9 years were occasional smokers and 1.5 percent were daily smokers. (Auth. Abs. Mod.)

84–1222. Nater, B.; Junod, B. **Evolution de la Consommation de Tabac Dans les Couples d'Adultes: Utilisation de la Methode de Knox. [Evolution of Tobacco Consumption Within Couples: Use of Knox's Method.]** *Sozial- und Praventivmedizin* 28(5): 238-239, 1983, French.

A means for investigating the interactive influences between members of a couple on tobacco consumption is outlined. The technique is an application of a method originally introduced by Knox to detect space-time clustering. When applied as part of a cardiovascular risk factor modification program in four Swiss cities, it was shown that the influence of family members in both increasing and decreasing tobacco use is significant.

84–1223. Nil, R.; Buzzi, R.; Baettig, K. **Personalichkeit, Typ A/B-Verhalten und Lebensgewohnheiten bei Rauchern und Nichtrauchern. [Personality, Type A/B-Behavior and Lifestyle in Smokers and Nonsmokers.]** *Sozial- und Praventivmedizin* 28: 242-243, May 1983, German.

In a comparison of smokers and nonsmokers (total, 421 healthy subjects), questionnaire results found differences in personality scores only in women, for openness. Across lifestyle variables, coffee consumption was higher in both sexes among smokers. In addition, among male smokers, the frequency of participation in sports and healthy eating habits were lower. Among women, alcohol consumption and the feeling of being weighed down by problems was higher in smokers compared with nonsmokers; vacation planning was lower. No differences were found in the six components of type A/B behavior.

84–1224. Parkes, K. R. **Smoking as a Moderator of the Relationship Between Affective State and Absence From Work.** *Journal of Applied Psychology* 68(4): 698-708, November 1983.

The hypothesis that smoking, and more specifically, negative-affect smoking, acts as a moderator of the relationship between affective state and absence in work settings with smoking restrictions was investigated among 192 female student nurses (125 nonsmokers, 27 relaxation smokers, 40 stress smokers) at two hospitals. Prior to exposure to the work situation, levels of affective distress did not vary significantly between groups differing in smoking behavior. Multiple regression analysis showed overall significant differences in absence frequency between hospitals and between smokers and nonsmokers. There was also a significant interaction between smoking behavior and affective state; only among stress smokers did initial level of affective distress predict frequency of absence. Since the study was carried out in a setting in which smoking was not permitted during working hours, the findings show that withdrawal from work in direct relation to affective symptom levels occurs only among persons who habitually smoke in response to stress. (Auth. Abs. Mod.)

84–1225. Pedersen, N. L.; McClearn, G. E. **Factor Structure of Common Drug Usage.** *Drug and Alcohol Dependence* 12(1): 97-109, August 1983.

Analysis of lifetime tobacco use revealed that both American and Swedish never smokers drank less alcohol and drank excessively less often than ever smokers. Swedish never smokers drank less coffee and tea than ever smokers; American never smokers used fewer sedatives than ever smokers. Analysis of current tobacco use showed that American smokers used more alcohol and sedatives than never or ex-smokers (the latter two groups used similar amounts). Among Swedes, never smokers used less alcohol than ex-smokers, and ex-smokers less than current smokers. Both never and ex-smokers used less coffee and tea than current smokers. The correlated usage of commonly employed, legal drugs was examined in 377 American and 908 Swedish adults. Measures of alcohol, tranquilizer, sleeping pill, and coffee or tea consumption were submitted to a principal components factor analysis with Varimax rotation. The resultant three factors were characterized by (1) heavy drinking in general and beer consumption specifically (ALCFAC); (2) use of tranquilizers and sleeping medications (TSFAC); and (3) consumption of coffee and tea (CTFAC). Factor structure profiles for ALCFAC and TSFAC were more stable than CTFAC across nationalities, sexes, and cohorts. Profiles for Swedish smokers and never smokers were very similar; for Americans, however, profiles for never smokers were more similar to Swedish profiles than to those of American smokers. Factor scores were computed to examine the relationship between tobacco use and levels of the factors by means of analysis of variance. In Swedes, ALCFAC and CTFAC levels varied with smoking status, whereas ALCFAC and TSFAC levels varied with smoking status in Americans. (Auth. Abs. Mod.)

84–1226. Pershagen, G. **Environmental Tobacco Smoke. 2.3. Validity of Questionnaire Data on Smoking and Other Exposures, With Special Reference to Environmental Tobacco Smoke.** *European Journal of Respiratory Diseases* 65(Supplement 133): 76-80, 1984.

Analysis of available data indicates that questionnaires can accurately discriminate between smokers and nonsmokers with regard to both respondents' and relatives' smoking habits. While accuracy is somewhat lower for detailed smoking habits of relatives, the information could also be useful in epidemiological studies. However, no data are available regarding the validity of environmental tobacco smoke exposure information, and the quality of such information is a suggested topic for future research.

84–1227. Petitti, D. B.; Friedman, G. D. **Evidence for Compensation in Smokers of Low Yield Cigarettes.** *International Journal of Epidemiology* 12(4): 487-489, December 1983.

In men and women of all ages, smokers of low-yield cigarettes smoked significantly more cigarettes/day than smokers of high-yield cigarettes. To determine the relationship of smoking low-yield cigarettes (tar yield <15.0 mg, and nicotine yield <1.0 mg/cigarette) with consumption levels, data were analyzed from self-administered questionnaires completed by 7,706 current, regular smokers. Mean age at smoking onset and mean number of smoking years were not consistently different in smokers of low-yield and high-yield cigarettes. Differences in number of cigarettes smoked/day between the two groups were small (about three cigarettes/day in men and about 1.5 cigarettes/day in women). These small differences, however, might translate into as many as 1 billion more packs/year in the United States alone. Any potential beneficial effect of smoking low-yield cigarettes must therefore be weighed against any possible adverse effects of increased consumption. (Auth. Abs. Mod.)

84–1228. Poewe, W.; Gerstenbrand, F.; Ransmayr, G.; Plorer, S. **Premorbid Personality of Parkinson Patients.** *Journal of Neural Transmission* Supplement 19: 215-224, 1933.

Preliminary findings from a study of 28 Parkinsonian patients indicate characteristic premorbid personality features that may correlate with previous observations of distinctive drinking and smoking habits in this patient group Parkinsonians premorbidly tend to be nonsmokers and teetotalers, behaviors that may be associated with an introverted personality type. The 14 male and 14 female patients (mean age 67 years; mean disease duration 9 years) were tested with the Giessen Test (GT) and patient/family interviews were conducted to obtain a retrospective evaluation of premorbid habits and traits. The most striking deviations from the mean GT score of a standard population were found in scales 3 and 4 for "control" and "basic mood," respectively. Patients strongly exhibited an overcontrolled personality type characterized by introverted and anancastic habits, compared with the impulsive, extroverted behaviors that have been noted in many studies of adult smokers. A significant deviation toward depression was also evident in females, but not in males. Premorbid personality assessments by patients and close family members further support these findings.

84–1229. Rose, J. E.; Jarvik, M. E.; Ananda, S. **Nicotine Preference Increases After Cigarette Deprivation.** *Pharmacology Biochemistry and Behavior* 20(1): 55-58, January 1984.

Nicotine preference, measured by the use of a smoke-mixing device, was significantly higher after 2 hours deprivation, indicating that nicotine is positively reinforcing. In determining whether cigarette smoking has a nicotine-seeking component, 20 smokers (12 males, 8 females; average consumption 29 cigarettes/day) were studied under two conditions: (1) deprivation (2 hours); and (2) satiation (smoking two cigarettes in 20 minutes). Subjects then used the mixing device to blend smoke with each puff from the high- and low-nicotine research cigarettes. Paired t-tests were used to compared puffing, nicotine preference, and ratings of strength and desirability between experimental conditions. Nicotine preference was more affected by deprivation in some subjects than others, apparently correlated with different subject characteristics, suggesting that different types of reinforcement are gained from smoking. Subjective judgments of strength and desirability were also affected, suggesting that smoking deprivation induces a substantial downward shift in the sensitivity to nicotine.

84–1230. Seiler, E. R. **Smoking Habits of Doctors and Their Spouses in South East Scotland.** *Journal of the Royal College of General Practitioners* 33(254): 598, September 1983.

Responses from 607 general practitioners in southeast Scotland (81 percent of those surveyed) revealed that 19 percent are smokers, but that only 6 percent smoke cigarettes, the main cause of morbidity. Ex-smokers account for 35 percent of the sample, yielding a total nonsmoking population of 81 percent. Among spouses of smoking physicians, 47 percent are also smokers, but only 9 percent of the spouses of nonsmoking doctors smoke. Findings suggest that general practitioners in southeast Scotland do not smoke cigarettes because the habit is harmful to health. A sponsored cessation campaign is recommended for those who continue to smoke.

84–1231. Shirlow, M. J. **Patterns of Caffeine Consumption.** *Human Nutrition: Applied Nutrition* 37A: 307-313, 1983.

Smoking was significantly correlated with caffeine consumption in a survey of 4,757 Australians that took into account individual variations in type and strength of coffee consumed. Average caffeine consumption was 240 mg/day, equivalent to approximately five 150 ml cups of medium-strength instant coffee. One-third of the population had intakes at this level or higher and may be considered to be physically dependent on caffeine. Correlation coefficients of smoking to caffeine, coffee, and tea were 0.17, 0.16, and 0.03, respectively. Of the respondents who smoked ≥ 30 cigarettes/day, 48.9 percent consumed ≥ 300 mg caffeine/day, while 43 percent took in ≤ 50 mg/day. Among nonsmokers, 19.6 percent consumed ≥ 300 mg/day, and 7.3 percent drank ≤ 50 mg caffeine/day. Only 3 percent of the population were true abstainers from caffeine. Consumption patterns were similar for men and women of all ages, although younger persons consumed more coffee and less tea than older persons. (Auth. Abs. Mod.)

84–1232. Swenson, I.; Dalton, J. A. **A Comparison of the Knowledge and Attitudes About Smoking Among Nurses Who Never Smoked, Those Who Have Successfully Stopped Smoking and Those Who Currently Smoke.** *International Journal of Nursing Studies* 20(3): 163-170, 1983.

Of 601 registered nurses in North Carolina who returned questionnaires (46 percent response rate), 321 (53.4 percent) had never smoked, 87 had successfully quit for 1 year, and 193 had smoked during the last year. While there were no significant differences in education, income, marital status, or working status between the groups, significant differences were observed between the never, former, and current smokers in their perception of smoking as a cause of illness. Of the current smokers, 53 percent felt smoking was important in health problems compared with 75.3 percent of former smokers and 78.7 percent of never smokers. The groups did not differ, however, in their agreement with the literature documenting the adverse effects of smoking. Concerning smoking in public places, 21.7 percent of ex-smokers vs. 7 percent of the current smokers had smoked in public while 54.7 percent of the ex-smokers, 55.4 percent of never smokers, and 42 percent of current smokers thought it unacceptable for nurses to smoke in public. Also, 5.8 percent of former smokers and 48 percent of current smokers disagreed with campaigns to restrict smoking in public places. More former smokers had participated in antismoking education programs than current or never smokers.

84–1233. Tabak Journal International. **How Britain Works to Make Pipe Smoking Youthful.** *Tabak Journal International* 6: 440-441, December 1983.

Mild, aromatic, and sometimes premium-priced pipe tobaccos are in fashion in Britain and selling well, although the overall decline in consumption has not yet been halted. The main target of sales drives is the young executive type of smoker, who prefers the delicacy of the new-style tobaccos. The industry is currently conducting multifaceted promotions featuring a youthful image and new pack styling. Pipe tobacco sales are also being encouraged by the health concerns over cigarettes and the pipe's tradition of social respectability. Two obstacles to the conversion of young smokers to the pipe are cited: the pace of executive life is not well suited to contemplative pipe smoking, and the trim clothing of younger males has no room for concealing the apparatus of pipe smoking. (Auth. Abs. Mod.)

84–1234. Treguer, M.; D'Houtaud, A.; Deschamps, J. P.; Martin, J.; Andre, G.; Chau, N. **Donnees Epidemiologiques Concernant le Tabagisme Chez les Jeunes. [Epidemiologic Data on Smoking Among Youth.]** *Medecine et Armees* 11(7): 595-600, October-November 1983, French.

Questionnaire surveys administered to French military recruits (average age 19.5 years) in December (n = 1,014) and June (n = 1,053) 1979 indicated that 59.8 and 66.7 percent, respectively, were smokers. Percentages of deferred students and ROTC candidates were higher in the December sample than the June sample (28 and 22.5 percent compared with 19 and 0 percent, respectively). The samples showed a parallel decrease in daily cigarette consumption and the ratio of subjects inhaling tobacco smoke. Observed differences in smoking habits between the groups were attributed to differences in sample composition. Responses to questions regarding parental and grandfathers' smoking habits were similar in both groups.

84–1235. von Pfeil, H. G. **Consumption of Quality Cigarettes Is Increasing in South Africa.** *Tabak Journal International* 6:478-479, December 1983.

Cigarette consumption is increasing in South Africa, partly at the expense of pipe tobacco, and the industry expects favorable rates of growth in the medium term. Although more people between 40 and 50 years of age are giving up smoking, the increase in cigarette use among younger groups more than compensates for lost sales. Between 1980 and 1982, cigarette consumption rose from 26,455 to 29,568 million kilos, while consumption of cigarette tobacco declined from 9,559 to 8,562 million kilos, owing to the trend toward filtered cigarettes. Cigar sales are also rising, while chewing tobacco and pipe tobacco continue to decline. It is noted that among the 10 new brands that have appeared on the South African market in the past 12 months, 2 are menthol cigarettes and the others are mild or ultra-mild brands. (Auth. Abs. Mod.)

84–1236. World Health Organization. **Guidelines for the Conduct of Tobacco-Smoking Surveys Among Health Professionals.** Geneva, World Health Organization, Document No. WHO/SMO/84.1, 1984, 19 pp.

A 10-page sample questionnaire for smoking surveys among health professionals, and guidelines for its use, are presented. The purpose of the document is twofold: (1) to encourage the conduct of such smoking surveys in countries where the information is not yet available, and to provide guidelines for planning such surveys; and (2) to standardize the data collection instrument as much as is feasible for the sake of comparability of different surveys within and between countries. Specific methods and procedures addressed are sampling, response rate and estimation, cross-sectional studies, special studies, self-administered questionnaires, selection of items to be included in the survey, data processing and presentation, smoking habit categories, confidentiality issues, and the constitution of the task force.

See also, **84–1090, 84–1162**

SMOKING PREVENTION AND INTERVENTION

84–1237. Catford, J.; Nutbeam, D.; Woolaway, M. C. **Giving Up Smoking—Are We Giving Up? (Letter).** *British Medical Journal* 287(6402): 1375-1376, November 5, 1983.

The deemphasis of the mass media in Britain as a means of promoting smoking cessation is deplored, noting the success of the Australian mass media program "Quit for Life" recently described (*British Medical Journal* 287(6399): 1125-1128, October 15, 1983). In Britain, which has the highest prevalence of smoking-related disease in the world, past efforts have been isolated and without ongoing support at the primary care level. Broad-based programs (including media, professional, and community action) are justifiable because (1) they work, as already demonstrated; (2) they are highly cost-effective; (3) most smokers want help in quitting; and (4) the general public (smokers and nonsmokers) wants the government to spend more to encourage people to stop smoking. While doctors now generally accept their role in prevention, they are largely unsupported, with little coordination of efforts at the district or national level. It is hoped that the new Chief Medical Officer can inspire a new, urgently needed initiative against smoking. See also 84-0607.

84–1238. Chapman, S. **The Lung Goodbye. A Manual of Tactics for Counteracting the Tobacco Industry in the 1980s.** Chippendale, Australian Consumers' Association, July 1983, 59 pp.

Prepared for the Fifth World Conference on Smoking and Health, the present manual outlines ways to frustrate the tobacco industry's marketing efforts, and means for shaping public and political opinion toward smoking control goals. Arguments and suggestions are included concerning how smoking control issues may be more forcefully politicized, thus giving politicians an imperative to act. The social aspects of the smoking issue are discussed, including (1) tobacco same-name products for children, (2) the puritan image of antismoking activists, (3) the tobacco industry's use of opinion surveys, and (4) toxic components of cigarettes. Industry arguments used to counter health-related arguments and defend tobacco products are covered. Finally, smoking control tactics are outlined, including activities such as forming independent groups, disrupting tobacco promotions, subverting sport and cultural sponsorship, political letterwriting, and infiltration.

84–1239. Chapman, S. **Effectiveness and Efficiency in Smoking Cessation Policy.** In: Carvolth, R. (Editor). *National Drug Institute: Innovations.* Proceedings of the National Drug Institute, Brisbane, Australia, 1983. Canberra, Australian Foundation on Alcoholism and Drug Dependence, 1983, pp. 11-14.

Smoking is a mass phenomenon in Australia, where an estimated 40 percent of males and 31 percent of females over the age of 16 are smokers. Most available smoking cessation programs are based on individual (behavior modification, psychotherapy, and hypnotherapy) and small group techniques that, from a policy standpoint, are labor intensive and costly. In responding to a referral request, therefore, a self-directed, individual effort should be recommended. When group therapy appears necessary, existing infrastructures should be used, and programs should facilitate contact with large numbers of smokers and should emphasize self-help. Expensive private programs should be avoided, while self-funding groups should be encouraged. Unfortunately, all existing programs are inconsequential in achieving significant increases in quit rates. An intensive effort is needed to identify features of effective approaches and incorporate them into a permanent mass media system that emphasizes prevention (especially among children), support for ex-smokers, and sound cessation strategies.

84–1240. Charlton, A. **Clues to Planning Smoking Education for the Sixteen-plus Age Group on Vocational Courses.** *Health Education Journal* 42(3): 71-73, 1983.

Age, sex, and type of vocational program had a marked effect on smoking habits among 153 boys and 280 girls surveyed as first year students at 16 colleges in England and Wales. Most of the students (33.5 percent smokers) were 16 and 17 years of age, but because they cannot be considered schoolchildren, and are not as old as medical students, student teachers, or student nurses, little information has been available about their smoking habits. Data show a progressive decrease in percentage of occasional smokers with age; in the oldest group, there were no occasional smokers. Although more girls than boys reported smoking (35.0 percent vs. 30.7 percent), many more girls (13.2 percent) than boys (3.3 percent) were only occasional smokers. In addition, boys who did smoke were heavier smokers. Marked differences were also found for type of vocational course: students of engineering and catering were least likely to be smokers (22 percent), while hairdressing and prenursing had the highest proportion (48 and 42 percent, respectively). It is concluded that smoking education must be tailored to each target group, and a followup study is planned that may provide keys to strategies for the various groups.

84–1241. Glasgow, R. E.; Klesges, R. C.; Godding, P. R.; Vasey, M. W.; O'Neill, H. K. **Evaluation of a Worksite-Controlled Smoking Program.** *Journal of Consulting and Clinical Psychology* 52(1): 137-138, February 1984.

The relative effectiveness of three versions of a controlled smoking program conducted in the worksite (abrupt reduction, gradual reduction, or gradual reduction plus feedback on nicotine consumption), was evaluated. All conditions were effective in producing reductions in each of three target behaviors (nicotine content, percentage of the cigarette smoked, and number). These reductions, as well as carbon monoxide changes, were maintained fairly well at a 6-month followup. There was some indication that the gradual reduction condition was more effective than the abrupt condition in assisting subjects to stop smoking. The addition of feedback on nicotine intake did not affect outcome. (Auth. Abs.)

84–1242. Morgan, P. P. **Toronto Department of Public Health Plans Community-Wide "Breakaway" From Smoking.** *Journal de l'Association Medicale Canadienne* 130(4): 358-359, February 15, 1984.

A 20-year smoking prevention plan for children has begun in Toronto, where 10 to 12 percent of seventh and eighth graders smoke. The program, titled "Breakaway," will teach children to resist peer pressure to smoke, and will also involve parents. In addition to the school program, businesses, health professionals, and community groups will be invited to participate in the stop smoking crusade. Toronto's success in the January 1983 "cold turkey challenge" against Vancouver, where 25 percent of those who pledged to quit were still abstinent after 4 months, led to the belief that a broad antismoking campaign was appropriate. During the first 3 years of the current campaign, financed by the city and headed by the Public Health Department, careful evaluations will be done to assist other cities in setting up programs. After the initial period, the project will be funded by community groups and agencies.

84–1243. Muller, M. **Preventing Tomorrow's Epidemic. The Control of Smoking and Tobacco Production in Developing Countries.** *New York State Journal of Medicine* 83(13): 1304-1309, December 1983.

Tobacco competes with other agricultural crops for available land, labor force, capital lent to farmers on a short- or medium-term basis, and energy required for curing. However, tobacco also enjoys a stable market and financial and technical support from the multinationals. Thus, farmers may opt to grow tobacco even if it is not the most profitable crop, while governments encourage tobacco production for its export value. For food crops to be encouraged, governments must stop investing national resources in tobacco. The future of profitable tobacco exports for Third World countries is questionable, since the technology available to farmers in industrialized countries will soon make it possible for them to compete equally with Third World farmers. Ultimately, cutting the demand for tobacco must be the focus of health advocates; both health and governmental revenue needs can be met by raising taxes, thus reducing consumption and raising funds without production investment. The implications of national tobacco monopolies in this process are discussed.

84–1244. Nares, P. **Colombia Restricts Home Smoking While Promoting Tobacco Exports.** *Tobacco International* 185(26): 84, December 23, 1983.

Delegates to the first Colombian Conference on Cigarettes and Health, sponsored by the State National Cancer Institute and the International Union Against Cancer, have recommended a broad-based antismoking campaign. Proposals include a ban on the sale of cigarettes to minors, restrictions on tobacco advertising, a ban on smoking in public places, measures to limit the tar content of domestic cigarettes, and higher tobacco taxes. Problems facing the antismoking campaign include the potential loss of tax revenues and employment benefits accruing from the tobacco industry. In addition, the government may leave itself open to charges of hypocrisy since it currently exports about 15,000 tons of black tobacco, valued at 32 million dollars, to Europe and North Africa. The two recommendations most likely to be adopted are the proposed increase in cigarette taxes and the call for tighter restrictions on advertising.

84–1245. South African Medical Journal. **Smoking — Changes in Emphasis.** *South African Medical Journal* 64(21): 803-804, November 12, 1983.

The Fifth World Conference on Smoking and Health participants recognized that the antismoking campaign is a war to be fought in the political arena, against the tobacco industry rather than the smoker. Control of cigarette use is now being seen in classic epidemiological terms: the whole chain of causation (social, economic, and political climates, and the marketing system of the tobacco industry) should be addressed, and not just the host (smoker). The medical profession cannot control the smoking habit, any more than it can control tuberculosis. It is noted that the most successful quitters usually do so of their own volition, while those who seek professional help are more apt to relapse. A total ban on tobacco promotion (not tobacco use) is proposed by the World Health Organization for all governments, as part of a comprehensive program. Such a ban would create an environment in which smoking is no longer perceived as socially acceptable, and in which health education can flourish free of opposition.

84–1246. Whyte, H. M.; Thurecht, L. **Stopping Smoking.** In: Carvolth, R. (Editor). *National Drug Institute: Innovations.* Proceedings of the National Drug Institute, Brisbane, Australia, 1983. Canberra, Australian Foundation on Alcoholism and Drug Dependence, 1983, pp. 51-52.

The outline of an intensive participatory workshop, conducted for providers of smoking cessation services in Australia, is presented. The workshop was divided into three sessions extending over about 5 hours. In the preparatory phase, aims, objectives, and agenda were outlined and participants introduced, followed by a general discussion. In the exploratory phase, a psychodramatic approach was utilized to illustrate the motivations for smoking (and persuasive environmental influences), and the negative, detractive aspects that must be utilized to influence smokers to alter their behavior. A final facilitatory phase examined approaches for intervening at various stages in the smoker's readiness for change and for effectively assisting the would-be quitter.

84–1247. Windsor, R. A.; Cutter, G.; Bartlett, E. E. **A Randomized Trial to Evaluate Health Education Interventions for Pregnant Smokers: Interventions, Methods, and Design.** Presented at the Fifth World Conference on Smoking and Health, Winnipeg, July 10-15, 1983, and at the Annual Meeting, American Public Health Association, Dallas, November 12-16, 1983, 35 pp.

A new 3-year prospective study of smoking cessation among pregnant women will document and validate smoking prevalence levels and quit rates at onset, midpoint, and last month of pregnancy among a large number of pregnant women randomly exposed or not exposed to cessation interventions. Two intervention groups, one given self-help skills education, the other a peer group support plan, will be compared with a control group. All groups will receive standard advice to stop smoking and drinking during pregnancy. It is estimated that a sample size of 113/group will be needed to confirm the statistical significance of observed quit rates and mean saliva thiocyanate changes. The maternal and infant health clinics involved registered 4,200 prospective mothers in 1982; 30 percent were smokers. Recent surveys indicate that from 75 to 80 percent of smokers would be willing to participate. It is expected that the rigorous methodology of the study (to be concluded in 1985) will provide valuable information.

84–1248. World Health Organization. **Smoking and Health. Global Medium-Term Programme. Programme 13.17.** Geneva, World Health Organization, Document No. NCD(SMO)/MTP/83.1, September 1983, 9 pp.

The World Health Organization's statement of policy and scope of the action program on smoking and health is presented. Within the context of the overall objective 13 of the Seventh General Program of Work, the objective of the Smoking and Health Program is to promote worldwide prevention and control of tobacco use, and of the diseases caused by it. Detailed in the policy statement are: (1) targets of the program; (2) approaches to the stated targets; (3) activities toward the development of national smoking control programs in all countries, training of health personnel and public education, and research; (4) program management and resources; (5) monitoring and evaluation of program effectiveness; and (6) global linking of already-existing smoking and health organizations.

SMOKING CESSATION METHODS

84–1249. Bobon, D. **Traitements de la Dependance au Tabac. [Treatment of Tobacco Dependency.]** *Revue Medicale de Liege* 38(17): 667-669, September 1, 1983, French.

While the reasons for cessation of smoking are well known (cost, health effects, esthetics, feeling dependent, social pressure, as example for others such as children, respect for nonsmokers), many studies have shown that motivation and continuing support (such as from a spouse) are the most important factors for successful cessation. The best way to quit smoking is "cold turkey," combined with any method available such as group therapy or acupuncture. Reduction techniques, which are not always successful, include switching from cigarettes to cigar or pipe, changing to low-nicotine, low-tar cigarettes, nicotine gum, nicotine-suppressing medication, and cigarette holders with filters. The 5-day group therapy method used in Liege is based on 10 commandments that include diet adjustment to exclude stimulants, consumption of 2 to 3 liters of water/day, physical activity, deep breathing, and early bedtime. While acupuncture appears to be helpful for some, results are no better than those of any other cessation method. Regardless of technique used, it has been shown that about 50 percent are abstinent at 6 weeks, 25 percent at 1 year, and after 5 years, 20 percent.

84–1250. Choy, D.S.J.; Lutzker, L.; Meltzer, L. **Effective Treatment for Smoking Cessation.** *American Journal of Medicine* 75: 1033-1036, December 1983.

During the use of tragus acupuncture to treat two cases of obesity in 1976, an unexpected side effect was discovered: the treatment also induced aversion to tobacco smoke and a sharply diminished desire to smoke. From May 1976 to December 1982, 514 patients were treated for smoking cessation using this approach. Of 339 evaluable patients, 297 stopped smoking, for a success rate of 88 percent. In a 2-year followup, the rate of relapse was 31 percent. Possible mechanisms of action were investigated with barium swallow, fluoroscopic determination of gastric and intestinal motility, and gastric emptying times with a technetium-labeled meal. No definitive conclusions could be drawn with respect to operative mechanisms. (Auth. Abs. Mod.)

84–1251. Labadie, J.-C.; Dones, J.-P.; Gachie, J.-P.; Freour, P.; Perchoc, S.; Huynh-Van-Thao, J.-P. **Desintoxication Tabagique: Acupuncture et Traitement Medical. Resultats Compares a 1 an sur 130 Cas.** [Detoxification From Nicotine Dependency: Acupuncture and Medical Treatment. Compared Results From 130 Cases After 1 Year.] *Gazette Medicale de France* 90(28): 2741-2747, September 30, 1983, French.

To compare the effectiveness of acupuncture versus medical treatment in curing nicotine dependency, 130 patients from a Bordeaux detoxification clinic were followed up for 1 year. Patients were randomly divided between the two treatments. Following initial consultation and examination, patients in the conventional group received a tranquilizer, a therapeutic nicotine substitute, and a detoxicant. In addition to treatment, acupuncture patients also received Tabacum 5 CH (to alleviate the desire to smoke), Nicogum (to keep the mouth busy), and Nux vomica tablets (to suck when nervous). Smoking cessation rates were nearly identical for the two groups at 1 year followup: 30.6 percent of the medical group and 31.5 percent of the acupuncture group quit smoking, while 11.4 percent and 16.1 percent decreased consumption, respectively. It was concluded that treatment is effective only when the patient is strongly motivated (the critical factor), in which case the prescribed method plays a supportive role.

84–1252. Russell, M.A.H.; Merriman, R.; Stapleton, J.; Taylor, W. **Effect of Nicotine Chewing Gum as an Adjunct to General Practitioners' Advice Against Smoking.** *British Medical Journal* 287(6407): 1782-1785, December 10, 1983.

Results indicate that it would be feasible and effective for general practitioners to include the offer of nicotine gum and brief instructions on its use as part of a minimal intervention routine with all cigarette smokers. Cigarette smokers (n=1,938) were assigned to either (1) nonintervention control group; (2) advice plus booklet; or (3) advice, booklet, and offer of nicotine gum. Subjects were followed up at 4 months and 1 year. There was a clear advantage for those using nicotine gum; the proportions abstinent at 1 year were 3.9, 4.1, and 8.8 percent in the three groups, respectively. Percentages include those who did not wish to quit and those in the gum group who did not try the gum (47 percent). The self-selected subgroup of 8 percent who used more than one box of gum achieved a success rate of 24 percent. A general practitioner who adopts such a routine with similar success could expect to achieve about 35 to 40 long-term ex-smokers a year (and save the lives of about 10 of them). If replicated by all general practitioners throughout the country, the yield of ex-smokers would be about one million/year. (Auth. Abs. Mod.)

84–1253. Sachs, D.P.L. **Office Strategies To Help Your Patients Stop Smoking.** *Journal of Respiratory Diseases* 5(2): 35-48, February 1984.

Current evidence suggests that if all physicians in the United States spent 60 seconds telling patients the health risks associated with cigarette smoking, more than 3.5 million smokers would quit who otherwise continue to smoke. Physician warnings to quit are especially effective in patients who already have symptoms of disease that might be caused or aggravated by smoking. Motivations for smoking range from nicotine dependence, which may be explained by the nicotine titration hypothesis, to psychologic dependence. Data from controlled, prospective, randomized clinical trials show that simple medical advice is probably the single most cost-effective therapy to help patients overcome the smoking habit. Effective cessation approaches must be based on sound pharmacologic-biologic-behavioral theory; e.g., nicotine chewing gum may be used alone, or in combination with behavior modification techniques such as rapid smoking therapy (in which cigarette smoke itself is the aversive stimulus). In selecting a treatment approach for individual patients, the Fagerstrom nicotine tolerance scale may prove useful in assessing the extent of nicotine dependency. Promising research on the role of β-endorphin in maintaining the smoking habit should yield new pharmacologic techniques during the next decade. (Auth. Abs. Mod.)

TOBACCO PRODUCT ADDITIVES

84–1254. Boden, R. M. **Smoking Tobacco Composition or Smoking Tobacco Article Containing 1-Ethoxy-1-Ethanol Acetate and Acetaldehyde.** United States Patent No. 4,421,125, December 20, 1983, 11 pp.

The uses of 1-ethoxy-1-ethanol acetate and acetaldehyde mixtures in flavoring tobacco are described. The compounds liberate an acetaldehyde nuance in tobacco, and also enhance its sweet, fruity, winey, fresh, and berry-like characteristics. A weight ratio of 1-ethoxy-1-ethanol acetate:acetaldehyde ranging from 50:50 to 5:95 is recommended, applied at a rate of 50 to 1,500 ppm of tobacco. The composition is stable and may be prepared by conventional processes.

84–1255. Boden, R. M. **Use in Flavors of Prins Reaction Products of Diisobutylene.** United States Patent No. 4,384,007, May 17, 1983, 10 pp.

The diisobutylene derivatives described (a mixture of dimers of isobutylene resulting from the Prins reaction) impart clove-like and minty taste and aroma nuances to tobacco smoking products. The flavoring mixtures are prepared by the dimerization of isobutylene in the presence of an alkylaluminum chloride catalyst. The mixture of dimers may be used as is, or further subjected to a Prins reaction with acetic anhydride and formaldehyde (or a formaldehyde source). The resulting acetates may be used as is, or further hydrolyzed and distilled.

84–1256. Boden, R. M. **3,4,5,6,6-Pentamethyl Hexanol-2 and Alkyl Homologues Thereof; Process for Preparing Same and Organoleptic Uses Thereof.** United States Patent No. 4,391,999, July 5, 1983, 27 pp.

A new compound genus of branched chain secondary alcohols is described, containing a substituted C_1-C_3 alkyl group at the R_1 position. The compounds are useful in augmenting or enhancing the aroma or taste of consumable materials including smoking tobacco, smoking tobacco articles, perfumes, colognes, and perfumed articles (including solid or liquid anionic, cationic, nonionic, or zwitterionic detergents). Tobacco is treated with 500 to 1,000 ppm of the secondary alcohol mixture, which imparts additional body and, on smoking, more Turkish-tobacco-like, aromatic, spicy, and woody/oriental aroma and taste nuances to both the mainstream and sidestream smoke.

84–1257. Boden, R. M. **Branched Chain Olefinic Alcohols, Thiols, Esters and Ethers, Organoleptic Uses Thereof, Processes for Preparing Same and Intermediates Therefor.** United States Patent No. 4,405,820, September 20, 1983, 35 pp.

Similar branched chain olefinic compounds and uses thereof for aroma and taste enhancement of tobacco products are described in 84–1259.

84–1258. Boden, R. M. **Branched Chain Olefinic Alcohols, Thiols, Esters and Ethers, Organoleptic Uses Thereof, Processes for Preparing Same and Intermediates Therefor.** United States Patent No. 4,399,062, August 16, 1983, 28 pp.

Similar branched chain olefinic compounds and uses thereof for aroma and taste enhancement of tobacco products are described in 84–1259.

84–1259. Boden, R. M. **Branched Chain Olefinic Alcohols, Thiols, Esters and Ethers, Organoleptic Uses Thereof, Processes for Preparing Same and Intermediates Therefor.** United States Patent No. 4,384,964, May 24, 1983, 60 pp.

Branched chain olefinic compounds with organoleptic properties are described. Compounds contain two C_1-C_4 alkyl substituted groups, and a substituted group at the R_3 position containing either oxygen or sulfur with hydrogen, a magnesium halide, lithium, a lower alkyl or a lower acyl. The uses of the novel compounds are as aroma and/or taste enhancers in smoking and chewing tobaccos, perfumes, perfumed articles (detergents and fabric softeners), chewing gums, medicinal products, and toothpastes. Processes for preparing the branched chain olefinic alcohols, thiols, esters, and ethers are described.

84–1260. Boden, R. M. **Branched Chain Olefinic Alcohols, Thiols, Esters and Esthers, Organoleptic Uses Thereof, Processes for Preparing Same and Intermediates Therefor.** United States Patent No. 4,375,005, February 22, 1983. 56 pp.

Similar branched chain olefinic compounds and uses thereof for aroma and taste enhancement of tobacco products are described in 84–1259.

84–1261. Boden, R. M. **Aliphatic Branched Olefin Dioxolanes, Dithiolanes, and Oxathiolanes and Uses Thereof in Augmenting or Enhancing the Aroma and/or Taste of Consumable Materials.** United States Patent No. 4,379,754, April 12, 1983, 54 pp.

A genus of compound is described that uses dioxolanes, oxathiolanes, and dithiolanes to provide specific sweet, woody, oriental, spicy, and fruity flavor characteristics of natural Turkish-like tobacco, prior to and on smoking, in the mainstream and sidestream smoke. Any convenient method for incorporating one or more of the described additives into the tobacco may be employed, and only a portion of the tobacco needs to be treated. The proportions of additives may be varied, but satisfactory results are obtained if the proportion to smoking tobacco material is between 50 ppm and 1,500 ppm (0.015 percent to 0.15 percent). Other consumable materials for which the additives are suitable as aroma and/or taste enhancers include foodstuffs, chewing gums, medicinal products, toothpastes, perfume compositions, and perfumed articles.

84–1262. Boden, R. M. **Prins Reaction Products of Diisobutylene, Derivatives Thereof, Organoleptic Uses Thereof and Processes for Preparing Same.** United States Patent No. 4,437,476, March 20, 1984, 20 pp.

The Prins reaction products of diisobutylene described provide oriental, fruity, rum-like, and Turkish tobacco notes to tobacco smoke. Concentrations of 250 ppm to 1,500 ppm of the active ingredients added to the smoking tobacco material are found to produce satisfactory results. The compounds are prepared by subjecting a dimer or dimers of isobutylene (prepared in the presence of alkylaluminum chloride catalysts) to a Prins reaction with acetic anhydride and either formaldehyde or a formaldehyde source in the presence of a Lewis acid.

The resulting mixture may be used as is or further hydrolyzed and added to the smoking material, the filter, or the leaf or paper wrapper by any convenient method. The products are also useful for flavoring other foodstuffs, chewing gum, perfumes, and medicine products.

84–1263. Boden, R. M.; Licciardello, M. **Flavoring With Cyclic Carbonate.** United States Patent No. 4,402,985, September 6, 1983, 13 pp.

The use of cyclic carbonate (4-isopropyl-5,5-dimethyl-m-dioxan-2-one) to improve and augment the organoleptic properties of smoking tobacco and smoking tobacco products is described. The cyclic carbonate in the tobacco or in the wrapper or filter is at levels of from 50 ppm up to 800 ppm based on the dry weight of the tobacco or the smoking tobacco article. The additive imparts, prior to smoking, sweet, vanilla, coumarinic, fruity, and cinnamon-bark-like aroma and taste nuances; and on smoking, sweet, fruity, and Virginia tobacco-like aroma and taste nuances. The cyclic carbonate can also be used to enhance the aroma and taste of other consumable materials.

84–1264. Bolt, A.J.N.; Chard, B. C. **Smoking Article.** United States Patent No. 4,434,804, March 6, 1984, 7 pp.

A method for adding a flavoring agent to the tobacco rod of a filtered cigarette is described, whereby the concentration of the agent is greater at the light-up end than at the region between the two end regions. The agent is added by causing a stream of air to pass through the fuel rod from the light-up end, causing the smoke-modifying agent to become entrained in the stream of air to form a particle cloud, and causing it to enter and pass along the fuel rod. The agent is introduced in such a manner that the amount released into the smoke varies as the article is smoked.

84–1265. Dahlgrun, R. **Method and Apparatus for Influencing the Permeability of Wrappers of Filter Cigarettes or the Like.** United States Patent No. 4,403,519, September 13, 1983, 16 pp.

A method and apparatus are described for preparing uniting bands used in a filter tipping machine to connect filter plugs to cigarettes, which allows a preselected quantity of cool air into the column of tobacco smoke when the cigarette is smoked. The bands consist of foraminous material, whose permeability can be varied by changing the quantity of adhesive applied when attaching the filter plug to the cigarette. The paster that applies adhesive to a continuous web about to be subdivided into uniting bands, is adjusted in response to signals generated by a testing device for the wrappers of successive filter cigarettes. If the permeability is too high, the paster is adjusted by reducing or increasing the quantity of adhesive applied to successive unit areas of the running web. This can be achieved by changing the thickness of the layer of adhesive or by changing the area of that portion of each unit length of the web coated with adhesive. (Auth. Abs. Mod.)

84–1266. Giersch, W.; Ohloff, G. **Unsaturated Alicyclic Ethers.** *United States Patent No. 4,396,781,* August 2, 1983, 5 pp.

Novel unsaturated alicyclic ethers and the process for preparing them are described. The compounds are useful as flavoring and perfuming ingredients. Two compounds, 1-methoxy-1,4,8-trimethyl-cyclodeca-4,8-diene and its ethoxy analog, confer on tobacco a woody, amber-like flavor like that of oriental tobacco. Compounds are prepared by reacting, in the presence of an acid catalyst, 2,5,9-trimethyl-cyclododeca-1,5,9-triene with a lower alkyl radical hydroxide.

84–1267. Grossman, H. **Cigarette and Filter for Tobacco Smoke.** United States Patent No. 4,396,026, August 2, 1983, 4 pp.

A filter for tobacco smoke is described that comprises a porous substrate that has a deposit thereon of particles that are crystalline, positively charged at the pH of tobacco smoke, water-insoluble, hydrophobic, and oleophilic to provide a coalescing agent, which acts to attract and neutralize the negatively charged particles that are dispersed in tobacco smoke. To increase the effectiveness of the filter, the tobacco has mixed therewith and deposited thereon particles of a water-insoluble, hydrophobic, moisture-laden, negative charge-imparting substance. The described filter, and cigarettes made with tobacco thus treated, act to remove a substantial amount of carcinogenic tars and nicotine without altering substantially the cigarette flavor. (Auth. Abs.)

84–1268. Hall, J. B.; Sanders, J. M.; Siano, J. N. **Process for the Production of Isosolanone and Solanone, Intermediates Useful in Said Process and Organoleptic Uses of Said Intermediates.** United States Patent No. 4,433,695, February 28, 1984, 44 pp.

A process for augmenting or enhancing the aroma or taste of a smoking tobacco or smoking tobacco article is described that provides various musty, oily, slightly green, tobacco-like, full-bodied burley tobacco aroma and taste notes. The additives contain as an active ingredient the 5-isopropyl-8-methyl-5,7-nonadien-2-one cis and trans isomer mix defined according to the process described. The additives may be used separately or with other flavor and aroma enhancers, and can be incorporated in the tobacco product by any convenient method. While particularly useful for smoking tobacco, other products formed from sheeted tobacco fines or dust may be treated, or the flavoring agent may be incorporated with filter tip materials, sham paste, or packaging materials.

84–1269. Klemarczyk, P. T.; Dekker, L.; Kiwala, J. **Substituted Methyl Isopropyl Cyclohexenones, Organoleptic Uses Thereof and Process for Preparing Same.** United States Patent No. 4,400,311, August 23, 1983, 61 pp.

A genus of novel compounds and the process for preparation of the compounds are described. The novel organoleptic compounds are isopropyl cyclohexenones containing substituted hydrogen, methyl, ethyl, 2-methyl-1-propenyl, or 2-methyl-1-propylidenyl groups, and are used in augmenting or enhancing the aroma or taste of consumable materials including smoking tobacco compositions, smoking tobacco articles, perfumes, and perfumed articles. Compounds are prepared by either dimerizing unsaturated ketones or by reaction of isobutyraldehyde and acetone or methyl ethyl ketone, each in the presence of an appropriate catalyst.

84–1270. Krasnobajew, V. **Microbiological Transformation Products of Beta-Ionone as Olfactory Agents.** United States Patent No. 4,402,989, September 6, 1983, 11 pp.

Novel odorant and/or flavorant mixtures prepared by fermentation of ionone type compounds by members of the genera Botryodiplodia, Botryosphaeria, or Lasiodiplodia are described. Medium requirements, culture conditions, and mass spectral data are provided. The products are said to improve the flavor of tobacco, foodstuffs, or drinks.

84–1271. Schreck, R. P.; Light, K. K.; Hall, J. B.; Schmitt, F. L.; Vock, M. H.; Schreiber, W. L.; Vinals, J. F.; Kiwala, J. **Alkyl Esters of 1-Alkanoyl Cycloalkanols and Organoleptic Uses Thereof.** United States Patent No. 4,374,055, February 15, 1983, 33 pp.

A process for preparing several substituted or unsubstituted cycloalkyl acyl alkanoates is described that provides compounds useful for their organoleptic properties as aroma enhancers of smoking tobacco, imparting sweet, minty, cooling, spicy, anisic, berry-like, fruity, pineapple-like, woody, green, pepper-like, and dill-like notes prior to and on smoking in the mainstream and sidestream smoke. In addition to one or more of the 1-acylcycloalkyl alkanoate derivatives, other flavoring and aroma additives may be added to the smoking tobacco materials, to the filter, or to the leaf or paper wrapper. Satisfactory results are obtained if the proportion by weight of the derivatives to smoking material is between 250 ppm and 1,500 ppm. The compounds produced are also useful as flavorants for foodstuffs, chewing gums, toothpastes, medicinal products, perfume compositions, and perfumed articles.

84–1272. Sprecker, M. A.; Hall, J. B. **Substituted Tricyclodecane Derivatives, Processes for Producing Same and Organoleptic Uses Thereof.** United States Patent No. 4,386,023, May 31, 1983, 49 pp.

The structures and preparation processes for a variety of substituted tricyclodecane derivatives having organoleptic properties useful for flavoring chewing and smoking tobaccos, and smoking tobacco products, are described. Also described are processes for using such derivatives and compositions containing them in perfumes, detergents, foodstuffs, chewing gums, toothpastes, cosmetic powders, and medicinal products. Smoking tobacco flavoring compositions and articles containing such compositions are also described.

84–1273. Willis, B. J.; Eilerman, R. G. **Substituted Cyclohexane Derivatives.** United States Patent No. 4,432,378, February 21, 1984, 17 pp.

When added to smoking tobacco, the substituted cyclohexanes described impart woody, amber-like, and cedarwood notes to the smoke aroma. Recommended proportions are 1 to 100 ppm. The flavorants are formed when the appropriate 2,4-dioxocyclohexane carboxylate is subjected to enol ether formation by treatment with a lower alkanol. The carboxylate derived is reacted with an olefin compound. The alkylated carboxylate formed is then decarboxylated by heating it in the presence of an alkali metal salt in a polar, aprotic solvent, yielding a cyclic ketone which is further transformed by one or a combination of steps to obtain the compounds of the invention.

84–1274. Willis, B. J.; Eilerman, R. G.; Yurecko, J. M., Jr. **Alicyclic Ketone and Alcohol Derivatives.** United States Patent No. 4,400,545, August 23, 1983, 11 pp.

The compounds described are useful as odor-modifying ingredients in tobacco products, foodstuffs, perfumes, and perfumed products. A 1 percent ethanol solution of 2,6-dimethyl-4-(3-methylbutyl)cyclohexanone was sprayed on a typical smoking tobacco in an amount sufficient to provide a tobacco composition containing 20 ppm of the flavor additive on a dry basis. Cigarettes were prepared from the treated tobacco and evaluated by a panel. Treated cigarettes were reported as having a light, woody taste, enhanced body, and fullness of flavor.

84–1275. Willis, B. J.; Fischetti, F., Jr.; Eilerman, R. G. **Flavoring Compositions and Flavored Comestibles Containing Alkyl 2,3-Dihydro-3(1′-Hydroxyalkylidene)-2-Oxo-5-Alkylfuran-4-Carboxylates.** United States Patent No. 4,382,972, May 10, 1983, 5 pp.

Composition of comestible flavoring agent containing alkyl 2,3-dihydro-3(1′-hydroxyalkylidene)-2-oxo-5-alkylfuran-4-carboxylates is described. For flavoring tobacco, a 1 percent ethanol solution of the ethyl derivative is sprayed on the tobacco and allowed to evaporate. The process may also be used on synthetic tobacco made of cellulose fibers. A striking improvement in flavor is reported.

TOBACCO MANUFACTURING AND PROCESSING

84–1276. Adams, J. M. **Variable Dilution Filter.** United States Patent No. 4,433,696, February 28, 1984, 6 pp.

A method is described for producing a cigarette wherein the smoke to ventilation air ratio may be changed to suit the preference of the smoker and that is compatible with current high speed cigarette manufacturing equipment. The cigarette is provided with a first stage filter that is rigidly attached to the tipping paper at the mouth end of the mouthpiece. A second stage filter element, located forward of the first stage element, is free to slide axially and may be made to move rearward by tapping the mouthpiece end of the smoking article on a hard surface. Rearward movement of the second stage filter element covers some of the ventilation holes located in the tipping paper between the first stage and second stage filter element, which increases the smoke to ventilation ratio, thus increasing the flavor of the cigarette.

84–1277. Cline, W. K.; Owens, W. F. **Wrapper for Smoking Articles and Method.** United States Patent No. 4,433,697, February 28, 1984, 5 pp.

A method is described for producing an improved magnesium oxide- and/or magnesium hydroxide-filled wrapping paper for smoking articles that incorporates at least 1 percent by weight of certain ceramic fibers that act to significantly reduce visible sidestream smoke. Using such wrappers, sidestream particulate matter is reduced at least 30 percent in smoking articles. The wrapper may be used as an inner wrapper under a normal outer wrapper of very porous or perforated cigarette paper or cigar wrap, or may be used as the single wrap for smoking articles. Wrappers may be made by incorporating the magnesium hydroxide, magnesium oxide, and other fillers in the wrapper pulp furnish containing the ceramic fibers, or the fillers may be applied to the improved wrapper as a coating for the papers. In the case of cigarette papers, the ceramic fibers may be added to an ordinary paper furnish such as pulped wood or flax fibers. The furnish of fiber pulp and ceramic fibers and fillers are used to make a paper sheet on conventional papermaking machines. (Auth. Abs. Mod.)

84–1278. Cornell, A. **The Current Status of Tobacco Leaf Expansion.** *Tabak Journal International* 6: 456, 458. December 1983.

Tobacco expansion is achieved by impregnating the cellular structure of the leaf with an agent capable of undergoing a rapid increase in volume on application of heat and/or reduction of pressure. Two major processes are used: the G-13 and the DIET (dry ice expanded tobacco) process. The G-13 process uses Freon and a high velocity mixture of hot gas under specific pressure and temperature conditions to expand the leaf. After expansion is completed, the Freon is recovered, condensed, and recycled. The DIET process involves soaking leaf in liquid carbon dioxide (CO_2), releasing the pressure, converting the liquid CO_2 to dry ice, then expanding the tobacco in a stream of hot gas. The CO_2 is not recovered. Other methods include the ammonium carbonate process, the INCOM method that uses gas phase nitrogen, and the HRV process that uses water as the expansion agent. Cost savings associated with increased filling capacity contribute to the use of expanded tobacco by the cigarette industry. Also, because less tobacco by weight is used, there is an added advantage of lower tar and nicotine delivery. (Auth. Abs. Mod.)

84–1279. Gaisch, H.; Ghiste, P.D.L.; Schulthess, D. **Process for the Preparation of Tobacco and Tobacco Prepared According to This Process.** United States Patent No. 4,407,307, October 4, 1983, 6 pp.

A process is described that provides tobacco whose content of protein components and subunits is considerably reduced, without reducing other soluble components. The insoluble proteins are initially made soluble by enzymatic treatment, dissolved, and then eliminated in the solution by metabolic assimilation. As a result of the metabolic assimilation, the undesired protein components and subunits, and low molecular weight nitrogen compounds such as amines, ammonia, nitrate and, if present, nitrite pass into the biomass, whereas the remaining soluble components that are to be dissolved out of the tobacco are essentially left behind in the residual solution and can be added again to the tobacco, optionally after evaporating the residual solution. (Auth. Abs. Mod.)

84–1280. Gillis, R. E. **Torus Smoking Pipe.** United States Patent No. 4,398,547, August 16, 1983, 3 pp.

A smoking pipe of unique design is described, which is in the form of an elongated or oval torus with a bowl at one end for the smoking material and a mouth hole at the opposite end. Twin passages extend from the bowl on either side of the torus to the mouth hole, so that there is a long path for smoke to travel and the smoke is cooled by air circulating between the two paths. The pipe is easily manufactured of materials such as ceramic, plastic, or wood. Variations in the exact structure can be made without departing from the basic principles of the design.

84–1281. Haber, W. **Cigarette Substitute.** United States Patent No. 4,429,703, February 7, 1984, 4 pp.

A cigarette substitute assembly is described that has the appearance of a cigarette in a cigarette holder. The assembly includes a plain hollow cylindrical tube with a mouthpiece at one end and a plastic member at the other end that resembles the ashes on a cigarette. The assembly also includes a cartridge that may be formed of porous material in a cylindrical shape and impregnated with an aromatic substance to give a pleasant smell and taste, simulating inhaling a mild menthol type cigarette, when the cartridge is mounted in the assembly and air is drawn through it. A silver-colored washer may be located between the mouthpiece and the end of the white plastic tube. The two end members have inwardly extending fingers that taper slightly to make a tight fit when assembled with the central tube and to grip and enclose the aromatic cartridge. The fingers may be provided with retaining protrusions to more securely hold the filter element in place, and each of these end members is provided with longitudinal passageways so that air may be drawn through the entire assembly. One end of the aromatic cartridge is therefore securely held by one of the end members; if desired, a resilient metal or plastic element, such as a coil spring of varying diameter, may be provided to hold the other end of the aromatic cartridge within the central plastic tube. (Auth. Abs.)

84–1282. Hibbitts, C. H. **Method for Expanding Tobacco With Steam at High Temperature and Velocity.** United States Patent No. 4,407,306, October 4, 1983, 4 pp.

A method for drying and expanding cut tobacco is described, which introduces the tobacco into an elongated, tubular-shaped conduit through which high temperature, high velocity, superheated steam is recycled. The conduit has a reduced area section into which the tobacco is introduced and provision is made for the recycling of steam, thus minimizing energy consumption. In the conduit, the tobacco's speed of travel never reaches the speed of the steam; thus, the surface of the tobacco shreds are constantly exposed to new, high temperature steam. The tobacco is expanded an acceptable amount without degradation or substantial diminution in size. (Auth. Abs. Mod.)

84–1283. Hinzmann, A. **Method and Apparatus for Monitoring the Locations of Perforations in Webs of Wrapping Material for Filter Cigarettes or the Like.** United States Patent No. 4,383,435, May 17, 1983, 15 pp.

A monitoring apparatus is described that allows testing of a rapidly running web of tipping paper for the size, presence or absence, and accuracy of location of ventilating perforations. The apparatus may also segregate defective products. Either pneumatic or photoelectronic devices are used to ascertain the distance between rows and the nearest marginal portions of the web while the web is transported toward the paster in a filter tipping machine. If the distance between the perforations and the marginal portion deviates from the desired distance, if the perforations are absent, and/or if the combined cross-sectional area of perforations per unit length of the web is unsatisfactory, the monitoring system generates signals that warn attendants, who then replace defective web or segregate any filter cigarettes embodying defective portions of the web. (Auth. Abs. Mod.)

84–1284. Joseph, H. D.; Koch, F. **Method and Apparatus for Monitoring the Functioning of Testing Devices in Machines for the Production and/or Processing of Smokers' Products.** United States Patent No. 4,403,620, September 13, 1983, 20 pp.

A method of monitoring the quality of filtered cigarettes is described in which the cigarettes, produced in a filter-tipping machine, are conveyed past several successive testing devices that monitor different characteristics of the cigarettes. The cigarettes then pass a removing device, a segregating device, and on to a conveyor for transport to a packing machine or into storage. When any of the testing devices detects a defective cigarette, it generates a defect signal that is transported to the segregating device for expulsion of the respective cigarette. The defective cigarettes can be conveyed only to and expelled by the removing device in response to the corresponding defect signals so that the removed cigarettes can be examined; or, the removing device can expel a given number of satisfactory cigarettes or one pair of successive cigarettes for examination. (Auth. Abs. Mod.)

84–1285. Lamb, C. G.; Sanford, R. A. **Cigarette Filter.** United States Patent No. 4,438,776, March 27, 1984, 4 pp.

A filter rod designed to both dilute smoke and increase the perception of smoke taste is described. A filter rod of porous material is wrapped with porous plug wrap and provided with longitudinal channels, which are open to the tobacco rod, but dead end prior to reaching the smoker's mouth. The channels at the periphery of the filter rod are longer than those at the center, with the shortest channel located at the rod axis. Because of differences in pressure drop between the channels, the velocity of the diluted smoke increases from the center of the filter rod radially outward. Thus, less diluted smoke enters the smoker's mouth in closer proximity to the taste buds, increasing the perceived taste to the smoker.

84–1286. Newton, R. P.; Harper, P. H.; Geiss, V. L.; Jewell, J. N. **Method of Processing Fresh Tobacco Leaves.** United States Patent No. 4,392,501, July 12, 1983, 6 pp.

A processing method for fresh tobacco leaves that results in a higher fill value, and which may allow for reduction of nicotine and nitrates, is described. Tobacco at 65 to 90 percent moisture is harvested and the lamina cut to appropriate size for use in a finished smoking product. The cut tobacco may then be subjected to microbial treatment to reduce nicotine, or various other extraction techniques. The cut leaf is then dried via freeze-drying, high absolute humidity drying, air-drying, or a combination of methods.

84–1287. Pezzi, G. **Method and Device for the Electropneumatic Testing of Cigarette Filters.** United States Patent No. 4,403,501, September 13, 1983, 7 pp.

A method and device are described for the electropneumatic testing of the air permeability of filter plugs for cigarettes to ensure that the permeability is between two preset thresholds (highest and lowest). Pneumatic pressure is applied at one end of the filter plug, while the other end of the filter cigarette is connected with the atmosphere through a bore. The pneumatic pressure applied at the inlet end of the cigarette plug is transformed into an electric test signal, which is compared with an upper alarm threshold and with a higher reject threshold, respectively, and with a lower alarm threshold and an inferior lower reject threshold. The test signal emitted upon reaching of the upper or lower alarm threshold initiates the operation of alarm indicators and/or sends a signal for the control of the filter plug producing machine, so as to modify the air permeability of the filter plugs. Upon reaching the upper or lower reject threshold, it causes rejection of the defective filter plugs. (Auth. Abs. Mod.)

84–1288. Shannon, J. F. **An Overview of Modern Tipping Materials for Cigarettes.** *Tobacco International* 186(1): 5-8, January 6, 1984.

Although true filter-tipped cigarettes were first produced in the 1940s, it was not until the 1950s that they made any impact on the cigarette market. Manufacturers became interested because they could sell the equivalent length filtered cigarettes cheaper since they contained less tobacco. In the 1960s, it was realized that perforating the tipping material (paper by then had replaced the original cork) was an effective way to reduce tar, nicotine, and carbon monoxide, important because of growing public concern about the health effects of smoking. The three basic perforating methods developed (mechanical, electrostatic, and laser) are described and advantages and disadvantages are discussed. The increasing demand for multicolor printing (replacing the traditional cork design), the need for papers designed specifically for perforating, and the running speeds of cigarette-making equipment resulted in a proliferation of grades used as base and tipping papers. Finally, the development of a heat-seal coated tipping process, which many felt was needed for high-speed processing (rather than the conventional water-based adhesive systems), is discussed.

84–1289. Taylor, G.W.B.; Barber, R. B. **Light Energy Perforation Apparatus and System.** United States Patent No. 4,404,454, September 13, 1983, 12 pp.

An improved apparatus and system are described for expeditious perforations of cigarette filter tipping paper by laser. The system provides an apparatus for generating pulsed light beams from a focused continuous laser beam and includes reflector discs that are rotated about a common rotational axis. The discs include peripheral reflective facets that confront the laser beam in the course of rotation, and also have light transmissive portions between adjacent facets. The facets and transmissive portions of successive discs are selectively misaligned so that pulsed beams issue successively from different discs. A system employing the apparatus for perforation of a web also includes a common lens per disc pair and a prism for deviating light issuing from one disc of the disc pair. (Auth. Abs. Mod.)

TOBACCO ECONOMICS

84–1290. Burry, R. D. **Cigarette Shipments, Leaf Tobacco Demand, Interest Expenses Falling.** *Tobacco International* 185(26): 6-11, 13-14, 16-17, December 23, 1983.

Each of the major domestic producers of cigarettes has reacted differently to a difficult 1983 earnings environment. Some companies have minimized net losses by gains in nontobacco enterprises, while others have slowed media expenditures, reduced interest expenses by divestiture and other cost-saving practices. The continuing trend, however, reflects previous shifts in industry fundamentals and argues against aggressive purchase of major tobacco equities in 1984. Estimates of near-term earnings by major product line for the four tobacco companies included in the Kidder, Peabody industry coverage (Philip Morris, R. J. Reynolds, American Brands, and U.S. Tobacco) are tabulated, followed by data on key determinants of near-term performance. These determinants include production and shipment trends, cigarette pricing at the producer, wholesaler, and retail levels, and raw material cost patterns.

84–1291. Cole, J. **Tobacco Alliance Fights Back.** *Tobacco Reporter* 111(2): 36-37, February 1984.

Although only recently organized (January 1983), the Tobacco Alliance (funded by the Tobacco Advisory Council; led by communications experts) has already displayed a vitality certain to establish it as a voice for the freedom to choose. To counteract the increasing publicity generated by antismoking activists, the Tobacco Alliance has drawn together groups and individuals whose livelihoods depended on tobacco, and has made them aware that (1) in countries where advertising has been totally banned, cigarette consumption remains unchanged; (2) if 40 percent of the adult population in the United Kingdom who are smokers were to quit, 11 million pounds/day in taxes would be lost; and (3) declining cigarette sales because of high taxes have put 9,500 tobacco workers out of work, and for each of these workers, 7 workers in related fields are also affected. Supporters, so far mostly persons directly involved in the manufacture of tobacco products, receive regular mailings of current information and are encouraged to recruit others. The alliance says it is "countering prejudice and ignorance with facts, cool arguments, and effective, coordinated action."

84–1292. Hall, R. **For Cigarette Marketers, Less May Be More in Age of Elastic Demand.** *Tobacco International* 185(26): 72, 74, 76, 78, December 23, 1983.

The price support freeze in the United States for cigarettes at 1982 levels has signaled a new era of elastic demand for both growers and users. The quantity of tobacco sold now depends more on the qualities and quantities needed to satisfy smokers than merely on competitive bidding for limited supplies. The freeze resulted from social, economic, and political changes over the past 20 years that altered the long-term elasticity of demand for cigarettes in relation to price changes. The reduced rate of growth in cigarette consumption that occurred from these changes led to a decreased need for cigarette tobacco and to conditions of inelastic demand and lowered revenues. Under the current price freeze, elastic demand conditions have been established, and a number of trends are anticipated by 1986: (1) growers, operating under a stable support price, will try to keep tobacco market prices stable; (2) manufacturers will purchase more tobacco for new product development than in the recent past; and (3) young adults, who have traditionally been heavy smokers, will be diverted toward use of illegal substances that are far more damaging to social well-being and health.

84–1293. Kling, F. **In West Germany Almost One Cigarette in Every Eight is Handmade.** *Tabak Journal International* 6: 500-502, December 1983.

During recent years, fine-cut tobaccos have enjoyed increased popularity in West Germany. In 1982, there were record sales of over 18,000 tons of such tobacco (a rise of 55.8 percent), corresponding to increases in hand-rolling or filling of cigarettes among West German smokers. Sales began increasing shortly after the unilateral price rise for cigarettes of about 5 percent in January 1982. In the second quarter of the year, traders stocked up with fine-cut tobaccos in preparation for the anticipated boom following an increase in the tobacco tax on June 1. Although this increase was also extraordinarily steep for fine-cut tobacco (about 150 percent), German consumers continued to turn to hand-rolling or filling cigarettes. It is estimated that in 1983 almost one cigarette in eight will be of that variety. The pipe tobacco market has become more stable after declining sales over recent decades, with the market continuing to offer a diverse range of products to suit the preferences of individual German smokers. (Auth. Abs. Mod.)

84–1294. Land, T. **Cigarette Advertising: Physicians Seek Stricter Controls.** *Tobacco Reporter* 111(2): 38, February 1984.

The British Medical Association (BMA) has called for strict legal controls on the indirect advertising of tobacco products (especially those that could influence young people to smoke), noting that a government study found that 20 percent of children between 11 and 16 years of age are smokers. The Advertising Standards Authority (ASA) does not include cigarette names and styles (in its ethical code), which are used to promote nontobacco products and activities. Significantly, teenage smokers tend to consume those brands most often seen in televised sports activities. The BMA says that the spirit of the code of advertising is "going up in flames," as the tobacco industry takes advantage of the loophole in an "insidious approach of concentrating on psychological imagery in which cigarettes in name are an essential part."

84–1295. Maxwell, J. C., Jr. **How the Brands Ranked. Maxwell International Estimates.** *World Tobacco* 83: 53, 55, December 1983.

Data are compiled for 13 countries, continuing the *World Tobacco* series on tobacco industry economic statistics for 1980, 1981, and 1982. Market shares are provided (for most countries) for filter vs. nonfilter cigarettes, and figures are listed according to leading brand position. Total sales are indicated. Information is tabulated for Bolivia, Colombia, Egypt, El Salvador, France, Malaysia, Panama, Peru, Philippines, Puerto Rico, Singapore, Turkey, and Uruguay. Only 1982 figures are given for Egypt. These data, combined with figures published in the June and September issues, cover most of the world's cigarette trade.

84–1296. Tobacco Institute. **The Tax Burden on Tobacco. Historical Compilation.** Washington, D.C., The Tobacco Institute, Volume 18, 1983, 198 pp.

The history of tobacco taxation in the United States is traced from 1865 to the present. Charts and tables provide data on Federal, State, and local taxes and collections; U.S. per capita consumption and sales; cigarette excise taxes compared with farm value of domestic leaf; and taxes as a percentage of retail price. Tax year highlights indicate that Federal, State, and municipal excise taxes on cigarettes for the fiscal year ending June 30, 1983, totaled more than 7.8 billion dollars (or almost 4 dollars per capita), an increase of 1.1 billion dollars over the previous year. The Federal excise tax, increased from 8 to 16 cents/pack on January 1, 1983, grossed over 3.4 billion dollars. State taxes yielded 4.2 billion dollars, an increase of 200 million dollars over 1982 collections, with highest taxes (26 cents) in Massachusetts and Connecticut. Municipal taxes grossed 174.4 million dollars. Federal and State taxes accounted for a nationwide average of 33.2 percent of the retail price of cigarettes, up from an average 26.8 percent in November 1982. The average retail price in November 1983 was 94.7 cents/pack (81.9 cents a year earlier), with average price/pack ranging from a low of 75 cents in North Carolina to a high of 1.13 dollars/pack in Connecticut. Over the past 120 years, tobacco taxes have generated revenues exceeding 155 billion dollars.

LEGISLATION

84–1297. ASHRAE Standards Project Committee. **ASHRAE Standard. Ventilation for Acceptable Indoor Air Quality.** American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, Georgia, 1981, 21 pp.

Ventilation requirements for acceptable indoor air quality in occupied spaces, as established by ASHRAE Standard 62-73 (revised), are based on a variety of factors, including the presence of smoking areas. Indoor air quality is considered acceptable if the required rates of acceptable outdoor air are provided for the occupied space and no unusual indoor air contaminants are present. Higher ventilation rates are specified for spaces where smoking is permitted because tobacco smoke is one of the most difficult contaminants to control at the source. Tabular data are provided on outdoor air requirements for ventilating commercial, institutional, residential, and industrial facilities. Expressed in terms of cubic feet/minute/person (cfm) or liters/second/person (L/s), these requirements often differ significantly for smoking and nonsmoking facilities, but can never be less than 5 cfm (2.5 L/s)/person regardless of the treatment of the recirculated air.

84–1298. Beardsley, T. **British Physicians Lose Patience.** *Nature* 306(5942): 418, December 1, 1983.

A recent report from the British Royal College of Physicians condemns promotional activities of American and British tobacco companies, stating that apathy and vested interest of government have prevented implementation of previous Royal College antismoking recommendations. Although reliable estimates of smoking-related deaths in Britain have reached at least 100,000/year, the Tobacco Advisory Council claims that there is no causal connection between smoking and disease, and that an advertising ban on cigarettes will not reduce consumption. The Royal College favors (1) an increase in the tobacco tax to allow tobacco companies to diversify and to prolong government tax revenues, (2) a ban on television advertising in the form of sponsorship of sports events, and (3) a reduction in yields of tar, nicotine, and carbon monoxide. The Royal College also opposes vigorous promotion by the tobacco industry in developing countries.

84–1299. Bjartveit, K. **Norway. Legislation and Political Activity.** *World Smoking and Health* 8(3): 13-22, Autumn 1983.

Restrictive measures that can be used to influence smoking behavior are listed, including advertising bans, health warnings and education, sales restrictions, product restrictions on tobacco content and emissions, taxation, and actual restrictions on smoking in public places and work areas. While imposing restrictions has been shown to be effective, it is necessary to observe public opinion before acting, to avoid a boomerang effect. However, surveys have shown a high percentage of the populations in Norway and Canada want stronger restrictions on smoking in public places, almost half of the Canadians want cigarette advertising eliminated, and 56 percent of the Norwegians support an increase in cigarette prices. Legislation without the necessary health education and cessation programs would probably be ineffective, however. Politicians must have the courage to pass legislation, even though it is not possible to quantify the effects, and be aware of the cynical attitudes of the tobacco industry, which has launched gigantic advertising campaigns in the Third World to replace markets being lost in the developed countries. If developed countries do not ban advertising, for example, it is not likely that the Third World will do so. The experience of Norway since the 1975 ban on advertising shows that smoking has declined in both sexes, and now corresponds with United Kingdom figures of more than 30 years ago. Norway is now working toward a smoke-free society by the year 2000. There will be political victims wherever smoking is raised as a political issue, through the power of the tobacco industry, suggesting that a worldwide political strategy, involving international organizations and the general public, will be needed.

84–1300. California, Assembly. **An Act to Add Section 25948 to the Health and Safety Code, Relating to Smoking.** Introduced in the California Legislature, 1983-1984 Regular Session, by Assembly Member La Follette, Assembly Bill No. 2997, February 14, 1984, 3 pp.

The legislative counsel's digest of Bill No. 2997, which adds a section to the California Health and Safety Code, is presented relating to establishment of nonsmoking areas in transportation services' waiting rooms. Section 1 of the bill outlines the role of tobacco smoke in contributing to indoor air pollution, health hazards to smokers, and the significant health hazards of involuntary (passive) smoking. Section 2 specifies the types of transportation services affected and provides that not less than 50 percent of waiting room areas be designated as no smoking areas. It also outlines the placement of signs to establish the area so designated. No reimbursement is provided, and the act does not contain a repealer (as noted in Section 3), and therefore, would remain in effect until amended or repealed by a later enacted act.

84–1301. Cox, H. T. **Smoking, Tobacco Promotion, and the Voluntary Agreements.** *British Medical Journal* 288(6413): 303-305, January 28, 1984.

The inadequacy of present legislation aimed at controlling tobacco advertising is illustrated by the proliferation of cigarette brand names on television via sports sponsorship. Yet successive British governments have spurned legislation and chosen voluntary agreements, which provide the tobacco industry with certain benefits: they preempt legislative measures; convert the Department of Health and Social Security from an executive health agency into an arbiter of conflicts between the health concerns and the tobacco marketers; allow the tobacco companies to function in some ways as a cartel; and have permitted the industry to restructure its advertising away from easily controlled media to the gray areas of sponsorship, adventure holidays, and clothing manufacture. Opponents of legislative control argue that (1) advertising directs attention to low-tar products, thus fulfilling an educational function; and (2) cigarette promotion does not influence levels of aggregate consumption. It is also noted that advertising serves to maintain the social acceptability of smoking, and plays a strategic part in rivalry between firms. Finally, given the continued volume of cigarette promotion, it cannot be claimed that bans would not lower consumption and that the voluntary agreements may ultimately make effective control more difficult.

84–1302. Iglehart, J. K. **Smoking and Public Policy.** *New England Journal of Medicine* 310(8): 539-544, February 23, 1984.

Twenty years after the release of the first Surgeon General's Report, the Government may be relinquishing its policy-balancing act between tobacco support and tobacco-related health concerns. Last July, the Senate Labor and Human Resources Committee reported a bill (Smoking Prevention and Health Education Act of 1983) that would strengthen the warning labels on cigarette packs and mandate disclosure of tar, nicotine, and carbon monoxide levels. The bill awaits floor action. A bill introduced in the House by Representative H. A. Waxman would require three different, strong warnings on cigarette packages and advertisements, placed on a random basis. The future of the House bill is in doubt, because various parties are searching for a compromise. Also, Congress will soon debate extension of the excise tax imposed in 1982, the first increase since 1951. The extra revenues may go to Medicare under a user-fee concept. It is noted that support for stronger labeling requirements is diverse, bipartisan, and unanimous among private health interests. Other branches of the Federal Government, such as the Civil Aeronautics Board and Federal Communications Commission, have also become involved. The American Medical Association, however, has chosen not to take a highly visible stance on the issue and has voted to withdraw from the National Interagency Council on Smoking and Health. In contrast, the Government's strongest stand yet was recently articulated in the release of the 1983 Surgeon General's Report, which is reviewed in detail, on the relationship between cardiovascular disease and smoking.

84–1303. New Jersey, Assembly. **An Act Controlling Smoking in Government Buildings and Supplementing Title 26 of the Revised Statutes.** Pre-Filed for Introduction in the 1984 Session by Assemblymen Herman, Pelly, Gill, Miller, Schwartz, Flynn, Weidel, Paterniti, Assemblywoman Perun, Assemblymen Haytaian, Doria, Albohn, and Deverin, Assembly No. 548, 1984, 5 pp.

An act controlling smoking in government buildings and supplementing Title 26 of the Revised Statutes protects both nonsmokers' interests and the smoker's right to smoke in designated areas. Supervisors must balance smokers' rights with the rights of nonsmokers to breathe clean air. Under terms of the bill, county and municipal health officials would enforce the law, which totally prohibits smoking in areas of government buildings open to public access. Provisions and penalties are defined.

84–1304. New Jersey, Senate. **An Act Controlling Smoking in Certain Restaurants and Supplementing Title 26 of the Revised Statutes.** Pre-Filed for Introduction in the 1984 Session by Senator Graves, Senate No. 639, 1984, 4 pp.

An act controlling smoking in certain restaurants and supplementing Title 26 of the Revised Statutes would establish the rights of nonsmokers to breathe clear air while not denying others the right to smoke. The bill would permit restaurants to establish smoke-free areas without requiring complex or onerous administrative or enforcement procedures. Restaurants seating 50 or more persons would be required to choose whether to maintain a nonsmoking area, notify the State Department of Health, and post signs accordingly. Enforcement would be by county and municipal health officials. Restaurants would not be responsible for enforcement provisions and would not be fined for failure to comply. Exceptions, provisions, and penalties are defined.

GENERAL

84–1305. Bell, J. **Government to Review Tar-Table Tests.** *New Scientist* 100(1384): 480, November 17, 1983.

Concern over the testing methods employed to produce published tar tables began in the United States, where it was claimed that Brown and Williamson's Actron filter (on the Barclay brand), produced less tar on a smoking machine than when smoked by a human. A U.S. appeals court subsequently ordered that all material claiming Barclay cigarettes to be 99 percent tar-free, or as having only 1 mg of tar, be destroyed. In response, the British Department of Health and the Laboratory of the Government Chemist are examining cigarette testing procedures to see if modifications are needed, and the Chairman of the Parliamentary Scientific Committee intends to invite scientists to give evidence about the way cigarettes are tested. Also, there is a campaign for a tobacco advertising ban, and a ban on the sale of Barclay cigarettes and the use of Actron filters has been requested, although a BAT spokesman claimed there was no known plan for marketing the cigarettes in Britain.

84–1306. Bell, J. **Smokers Make Mockery of Tar Tables.** *New Scientist* 100(1381): 251, October 27, 1983.

The compensatory practices of smokers, which serve to raise the tar and nicotine yields of cigarettes, are undermining the British Government's strategy on smoking and health. The cigarette companies have warned the Government not to push for a further reduction in the tar ratings of cigarettes for fear of encouraging compensatory smoking. The Scientific Committee on Smoking and Health (SCSH) has reported that the nicotine level in some cigarettes is at a point where smokers may compensate to maintain nicotine intake, and thus compromise previous recommendations to reduce lifetime smoke exposure. Although studies of compensatory smoking behavior have already been published, the SCSH would like to obtain results from widespread long-term research, if possible by the tobacco industry. However, such research might draw attention away from the real issue of cigarette advertising, just as Brown and Williamson are preparing to launch the Barclay brand (with its controversial Actron filter) in the United Kingdom.

84–1307. British Medical Journal. **Health or Smoking.** *British Medical Journal* 287(6405): 1570-1571, November 26, 1983.

Although there has been some decline in smoking rates, tobacco still accounts for 15 to 20 percent of all deaths in Britain, but the government refuses to do anything to stem the tide of new smokers among children and young adults. Of every 1,000 young male smokers, 1 will be murdered, 6 will die on the roads, and 250 will be killed by tobacco-related diseases. More recently, the effects of passive smoking on the health of nonsmokers have been scrutinized, and tobacco industry promotion activities in Third World countries have been condemned as clear evidence of callous indifference to health. Further progress in the control of smoking-related diseases must come through action by the British government, out of concern for the health of its own people and from an awareness of its role as an exemplar in developing countries.

84–1308. Cattelain, P. **Une Autre Maniere de Considerer le Tabagisme. [Another Way to Look at Smoking.]** *Revue Medicale de Liege* 38(17): 669-673, September 1, 1983, French.

The paradox surrounding the subject of smoking, with many publications devoted to the dangers while others report the possible high quality proteins that might be derived from tobacco, is discussed. Various researchers are quoted, who do not agree completely with the cited dangers of smoking, suggesting that (1) smoking could not be considered a vice or a sin; (2) sometimes smoking has a calming effect on the nervous system; and (3) the pleasurable as well as the harmful effects should be considered. It is recommended that experts get together to strike a balance between the social costs of smoking and any possible benefits. Steps taken by the Belgian tobacco industry to make smoking less of a problem are cited, including (1) support of scientific research at various universities and in their own laboratories; (2) restriction of promotion to minors so their choices will not be influenced; and (3) development of safer products, with reduced levels of known noxious substances, over the past 20 years.

84–1309. Dijkstra, B.K.S. **Smoking — A Different Viewpoint.** *South African Medical Journal* 65(3): 74, January 21, 1984.

Exception is taken to a recent editorial (*South African Medical Journal* 64(21): 803-804, November 2, 1983) calling for increased attention to the "smoking hypothesis." It is argued that data exonerating smoking as a possible cause of illness and death can be found in publications of leading medical practitioners and scientists. Findings from tests of 69 South African cigarette brands show extremely high tar and nicotine content, although the incidence of lung cancer in that country is among the lowest in the world. The editors reply that data recently released by the Royal College of Physicians provide an even stronger condemnation of tobacco. The British Government is urged to reverse its present conciliatory attitude toward the tobacco industry and to take urgent action against this serious public health hazard.

84–1310. Houston, T. P. **The Mythology of Low-Tar Cigarettes.** *Journal of the Medical Association of Georgia* 73. 93-94, February 1984.

The alleged safety of low-tar cigarettes is a myth, because smokers who switch to low-yield products tend to change their smoking habits to compensate for the reduction in nicotine intake. A variety of studies have also negated the idea that low-yield cigarettes are less harmful to health. Yet the tobacco industry continues to use deceptive promotion to keep people smoking supposedly safer products. e.g., the success of Brown and Williamson's new Barclay brand may be because its special filter delivers three to seven times more tar and nicotine than the advertisements claim. Also, a particular Carleton package, advertised as "ultra low," is actually only one of eight varieties; the other seven, which have higher yields than the advertised style, are the ones generally available at retail outlets. Physicians are encouraged to join with the American College of Chest Physicians in their comprehensive antismoking campaign.

84–1311. Leu, R. E.; Schaub, T. **Does Smoking Increase Medical Care Expenditure?** *Social Science and Medicine* 17(23): 1907-1914, 1983.

The impact of smoking on medical care expenditures is analyzed, challenging the widespread belief that smoking imposes a large cost burden on health services systems. The results imply that lifetime expenditure is higher for nonsmokers than for smokers because smokers' high annual utilization rates are overcompensated for by nonsmokers' higher life expectancy. Population simulation, taking into account the effects of past smoking on present population size and composition, suggests that 1976 expenditures would have been the same if no male born since 1876 had ever smoked. The male population would have been larger, particularly at older ages, increasing medical care expenditure, but this increase would have been offset by lower annual medical care utilization rates. Thus the results imply that smoking does not increase medical care expenditures and, therefore, reducing smoking is unlikely to decrease them. (Auth. Abs.)

84–1312. Leu, R. E.; Schaub, T.; Schmid, H. **Die Volkswirtschaftlichen Kosten des Rauchens: Eine Replik. [The Political-Economical Costs of Smoking: A Reply.] (Letters).** *Schweizerische Medizinische Wochenschrift* 113(38): 1367-1370, September 24, 1983, German.

The social and economic costs of smoking that were previously discussed (*Schweizerische Medizinische Wochenschrift* 113(1): 1-2, January 8, 1983; *Munchener Medizinische Wochenschrift* 125(8): 28, February 25, 1983) are reviewed, noting that estimates of costs for Switzerland may be lower than in other countries. Compared with the United States where the calculated costs in 1976 were about 28 billion dollars, at the same ratio, costs for Switzerland would be about 1.5 billion Swiss francs. Data from one American study found the incidence of smoking-related disease to be 83 percent of lung cancer, 67 percent of chronic bronchitis and emphysema, and 20 percent of coronary heart disease. Estimates of the direct costs of such diseases are placed at 8 percent of total health costs. The distribution of the socioeconomic costs of smoking (tobacco industry, taxation, and consumer-related items) in Switzerland are also reviewed. The original author notes that he had merely wanted to raise the problem of the economic significance of smoking, as a followup to other published information, to emphasize the complexity of the situation. See also 83–1456, 83–1706.

84–1313. New Scientist. **An Industry's Last Gasp.** *New Scientist* 100(1384): 474, November 17, 1983.

To ban cigarette promotions would save lives, yet the British government's stand on advertising has never been convincing. The health warnings are also inadequate, and recently questions have been asked as to the meaning of published tar yields. The introduction of Brown and Williamson's Actron filter, and the realization that smoking machines do not reflect actual human consumption, have discredited the concepts of high-, medium-, and low-tar categories. Besides, smokers who switch to low-tar brands may smoke more, thereby increasing tobacco industry profits and the government's tax income without curbing smoking-related disease. It is noted that few past or present members of Parliament believe tobacco promotions should be allowed or that the industry lives up to the Advertising Standards Authority's criteria of "legal, decent, honest, and truthful." Parliament is advised to abolish tobacco industry promotions, and not be distracted by the tax income it provides, or by the tar table issues.

84–1314. Sarfaty, G. **Smokescreen.** *Medical Journal of Australia* 2(5): 245, September 3, 1983.

Strong exception is taken to a full-page advertisement ("Let's be sensible about smoking"), sponsored by the Tobacco Institute of Australia Ltd., which appeared July 28, 1983, apparently motivated by the success of the Quit For Life campaign. Counterarguments to the Institute's claims are: (1) advertising does not affect cigarette consumption (why, then, spend 50 to 60 million dollars/year on advertising?); (2) a total ban on production does not reduce consumption (smoking rates declined from 16 to 13 percent for boys and from 16.5 to 11 percent for girls after Norway's ban); (3) cigarette smoking is not addictive (it is habituating); (4) passive smoking does not cause disease in healthy nonsmokers (evidence to the contrary exists); and (5) the whole case against smoking is statistical (cholera was abated in England with less statistical evidence). Thus, the New South Wales Cancer Council has exposed the Institute's advertisement as a "smokescreen."

84–1315. Sterling, T. D.; Sterling, E. M. **Environmental Tobacco Smoke. 1.2. Investigations on the Effect of Regulating Smoking on Levels of Indoor Pollution and on the Perception of Health and Comfort of Office Workers.** *European Journal of Respiratory Diseases* 65(Supplement 133): 17-32, 1984.

A review of data from sealed, energy-efficient buildings (some investigated because of illness complaints) concludes that neither pollution levels nor patterns of symptoms differ between buildings with or without smoking restrictions. Trace amounts of aromatic hydrocarbons were found in many offices, but average levels of pollutants did not differ between buildings where smoking is permitted and where it is restricted. Also, no differences were found between the buildings associated with illness complaints and those that were not. It is noted that employees working where smoking is permitted reported substantially less stress than those where smoking is restricted, although there was a strong tendency for a high percentage of nonsmokers to rank building environment as poor in workplaces where smoking is permitted. Overall, health-related complaints were unrelated to workplace smoking regulations, and it is concluded that under conditions of inadequate ventilation, discomfort and illness result whether or not cigarette smoke is present. Symptoms decline dramatically (a 31 percent decline for eye irritation alone) whenever ventilation is increased and/or high ultraviolet emitting lamps are replaced.

84–1316. Van Peenen, P.F.D.; Blanchard, A. G.; Wolkonsky, P. M. **Leanness and Smoking. (Letter).** *Journal of the American Medical Association* 250(17): 2284, November 4, 1983.

Confirmation of the relationship between weight and smoking found in male participants in the Framingham Heart Study (*Journal of the American Medical Association* 249(16): 2199-2203, April 22, 1983) is provided by data on 3,393 male oil refinery workers. Analyses of current smoking habits showed a consistent trend for increasing cigarette consumption with increasing leanness. Leanness was determined by Quetelet index; prevalence of smoking was analyzed by each component of the index (height and weight). Data showed that the smoking trend was related only to weight. See also 83-1105.

84–1317. Walsh, D. C. **Corporate Smoking Policies: A Review and an Analysis.** *Journal of Occupational Medicine* 26(1): 17-22, January 1984.

Available evidence on corporate smoking policies and programs is presented, the motives and considerations involved in employers' decisions are explored, and the place of such activities in the broader context of corporate concerns over health care costs are discussed. Basically, policies fall into two categories: (1) restrictions on smoking in the workplace or limiting certain jobs to nonsmokers; and (2) programs designed to encourage and assist employees in overcoming the smoking habit. Cessation programs may include physician's counseling, written materials on how to quit, posters, clinic sessions, and monetary incentives. Strategies employed to change smoking behavior (empirical—rational, normative—reeducative, and power—coercive) are described as they apply to company-based programs. While there are confounding factors that cloud the issue of cost vs. benefit, including the breadth and power of the tobacco industry, some companies are reassessing their position on smoking, for a variety of reasons, e.g., to anticipate demands of nonsmoking employees, who are now in the majority. The growing concern over company-borne health care costs also makes the cigarette smoking question of vital importance, as companies begin to identify high-cost users of health care.

AGRICULTURE

84–1318. Bowman, D. T.; Wernsman, E. A.; Corbin, T. C.; Tart, A. G. **Contribution of Genetics and Production Technology to Long-Term Yield and Quality Gains in Flue-Cured Tobacco.** *Tobacco International* 186(6): 84-89, March 16, 1984.

The relative contributions of flue-cured tobacco (*Nicotiana tabacum* L.) breeding and changes in production technology to improve yield and quality were assessed for the period from 1954 to 1981. Regression analyses were conducted for North Carolina Official Flue-Cured Tobacco Cultivar Tests (NC OVT), using years as an independent variable (X) and lead yields and quality for the long-term check cultivar "Hicks" as dependent (Y) variables. The least squares regression coefficient for Hicks was used to estimate changes resulting from improvements in production technology. Differences between the mean of new cultivars entered annually in the NC OVT and Hicks were calculated for yield and quality, and regressions of these differences were employed to estimate genetic contributions to tobacco improvement. An annual estimated yield increase of 49.5 kg/ha was observed, 68 percent attributed to improved production technology and 32 percent to genetic improvement. Quality as defined by physical appearance and measured in dollar value/100 kg showed significant improvement with an average annual increase of 26 cents/100 kg. Improved production technology has also significantly increased nicotine and nitrogen levels, but has had little effect on reducing sugars. (Auth. Abs.)

CHEMISTRY

84–1319. Coleman, R. F. **Smoking Tests. (Letter).** *New Scientist* 100(1382): 369, November 3, 1983.

In reply to previous comments (*New Scientist* 100(1382): 369, November 3, 1983), a representative of the Laboratory of the Government Chemist (London) defends the procedures used to determine tar levels of cigarettes. The procedures, which have been used for 13 years are considered relevant for the purposes intended: establish standardized figures for ranking cigarettes that demonstrate trends in reduction of tar yields over the years. It is stated that the current methodology, which now also provides data on carbon monoxide content, provides an adequate means for those wishing to choose between brands on the basis of tar, nicotine, and carbon monoxide yields.

84–1320. Gholse, S. B.; Deshmukh, B. K. **Nicotine Content of Some Chewing Tobacco.** *Indian Journal of Pharmaceutical Sciences* 45(2): 95-97, March-April 1983.

Tobacco chewing is common in India and East Asian countries, and chewers are often victims of various lung, mouth, and heart diseases caused by toxic constituents such as nicotine. Eight varieties of tobacco used by the people of the Nagpur (Vidarbha) region were analyzed for nicotine content by a spectrophotometric method with bromophenol red as the coloring reagent. The complex has an absorption maximum at 575 nm. Beer's law is obeyed between 0.08 and 4.0 μg/ml nicotine, while molar absorptivity is 1.75×10^4 cm^2/mole. Results show that the nicotine content in the eight brands of chewing tobacco varies from 0.14 to 0.89 percent. (Auth. Abs. Mod.)

84–1321. Ishizu, Y.; Ohta, K.; Tomita, H. **Physical Properties of Tobacco Smoke Particles Produced Under Different Conditions.** *Beitraege zur Tabakforschung International* 12(3): 137-146, February 1984.

Smoke particle weight increases when produced in an atmosphere with helium gas and a higher heating rate. Small samples of tobacco powder, prepared by grinding dried tobacco leaves, were heated in a microthermobalance in different atmospheres and at different heating rates. Size distribution and mass concentration of smoke particles produced were measured simultaneously with a laser particle counter and a piezo balance mass monitor. The change in weight loss with time was also measured during each experiment. It was found that a larger amount of smoke particles was produced when tobacco was heated in an atmosphere of inert gas and/or at higher heating rates. Comparison of measured and calculated size distributions showed that particle size distribution was governed mainly by coagulation. (Auth. Abs. Mod.)

84–1322. Leete, E. **The Methylation of Nornicotine to Nicotine, a Minor Biosynthetic Pathway in *Nicotiana tabacum*.** *Beitraege zur Tabakforschung International* 12(3): 113-116, February 1984.

While it was established that the methylation of nornicotine to nicotine does occur in the tobacco plant, it is not considered to be a major pathway for the biosynthesis of nicotine. Nornicotine is formed by the demethylation of nicotine; however, to determine if the reverse reaction occurs, (*RS*)-(2'-^{14}C)nornicotine was fed to *Nicotiana tabacum* plants. Most of the administered nornicotine was recovered unchanged, but a small amount of activity was detected in the nicotine.

Activity was also found in myosmine. Nicotine was degraded and found to have essentially all its radioactivity located at the C-2′ position, indicative of a direct synthesis from nornicotine. (Auth. Abs. Mod.)

84–1323. Pryor, W. A.; Uehara, K.; Church, D. F. **The Chemistry and Biochemistry of the Radicals in Cigarette Smoke: ESR Evidence for the Binding of the Tar Radical to DNA and Polynucleotides.** In: Bors, W.; Saran, M.; Tait, D. (Editors). *Oxygen Radicals in Chemistry and Biology.* New York, Walter de Gruyter and Company, 1984, pp. 193-201.

It is demonstrated that the free radical in cigarette tar binds to poly(G) and to DNA, suggesting a mechanism for smoking-related carcinogenesis. Four polynucleotides and calf thymus DNA were incubated with whole cigarette tar. After incubation, all four of the polynucleotides and the calf thymus DNA showed an ESR signal that was similar to the tar radical; the reisolated poly(G) and calf DNA gave significantly stronger ESR signals. The g-values of the ESR signals from the recovered substances were slightly larger than for radical D. These results suggest that the principal binding site is at guanosine residues and that the binding is due to the tar radical itself.

84–1324. Rickert, W. S.; Robinson, J. C.; Young, J. C.; Collishaw, N. E.; Bray, D. F. **A Comparison of the Yields of Tar, Nicotine, and Carbon Monoxide of 36 Brands of Canadian Cigarettes Tested Under Three Conditions.** *Preventive Medicine* 12(5): 682-694, September 1983.

Although the trend toward cigarettes with low average deliveries of toxic substances may result in reduced health risks for some people, switching to low-yield cigarettes may not reduce the risk for smokers who smoke these products intensively. Thirty-six brands of Canadian cigarettes, including 28 with ventilated filters, were tested under standard smoking machine conditions and two other conditions to determine how yields of tar, nicotine, and carbon monoxide change when cigarettes are manipulated to increase smoke intake. While the rank order yield of tar, nicotine, and carbon monoxide was preserved, the average yield of all three substances more than doubled when cigarettes were smoked intensively in comparison with standard smoking. Findings emphasize the need to consider the dependency of tar—nicotine ratios on smoking conditions when assessing the proposed advantages of a medium-nicotine, low-tar cigarette. (Auth. Abs. Mod.)

84–1325. Snook, M. E.; Chortyk, O. T. **The Rapid Determination of Harman and Norharman in Cigarette Smoke.** *Tobacco International* 186(7): 97-101, March 30, 1984.

Three methods of isolation and quantitation of harman and norharman from cigarette smoke have been developed and evaluated. After trapping by dry ice, solvents, or glass fiber filters, basic compounds were separated by aqueous acid extraction. The use of an internal standard and glass capillary gas chromatography showed that analyses were rapid, quantitative, and reproducible. Smoke condensates from 18 different cigarettes of varying tar levels were analyzed, and a linear relationship was found between tar levels and harman and norharman production. Different tobacco types or tobaccos with different alkaloid contents had no effect on harman and norharman production. (Auth. Abs. Mod.)

84–1326. U. S. Department of Health and Human Services. **Third Annual Report on Carcinogens. Summary.** U. S. Department of Health and Human Services, Public Health Service, NTIS No. PB-83-135855, September 1983, 234 pp.

Information from the second report is updated with 28 new entries. For each listing, available information is given on the nature of exposures, estimated number of persons exposed, and extent to which implementation of Federal regulations decreases the risk to public health. Substances are separated into 19 "known carcinogens" (a causal relationship has been demonstrated in human studies) and those "which may reasonably be anticipated to be carcinogens" (based on limited evidence from human studies or sufficient evidence in experimental animals). Of the 117 entries, 2 are natural substances, 2 are food and cosmetic additives, 12 are pesticides, 15 are therapeutic substances, and the remainder are various industrial chemicals and byproducts. Ionizing radiation, ultraviolet radiation (including sunlight), tobacco, alcoholic beverages, and some viruses are known or suspected to be carcinogenic, but have not been included because ionizing radiation is discussed in a General Accounting Office report, and a recent Surgeon General's report reviews the overwhelming relationship between tobacco and cancer. Several publications explain the relationship of alcoholic beverages, ultraviolet radiation, and viruses with cancer.

PHARMACOLOGY AND TOXICOLOGY

84–1327. Adriaenssens, P. I.; Sivarajah, K.; Boorman, G. A.; Eling, T. E.; Anderson, M. W. **Effect of Aspirin and Indomethacin on the Formation of Benzo(*a*)pyrene-Induced Pulmonary Adenomas and DNA Adducts in A/HeJ Mice.** *Cancer Research* 43(10): 4762-4767, October 1983.

Previous results in various in vitro systems suggest that prostaglandin endoperoxide synthetase (PES) could serve as either an alternative or an additional enzyme to the cytochrome P-450-dependent monooxygenases for the formation of mutagenic, cell-transforming, and DNA-binding metabolites of carcinogens. To test this hypothesis in vivo, the effect of PES inhibitors on benzo(*a*)pyrene (BaP)-induced pulmonary adenoma and BaP metabolite:DNA adduct formation was examined in A/HeJ mice. Animals were treated with a dosage regimen of aspirin that inhibited PES, but had no effect on the cytochrome P-450-dependent oxidation of 7,8-dihydrodihydroxybenzo(*a*)pyrene. Aspirin did not significantly alter the number of pulmonary adenomas per mouse at either dose of BaP (6.0 and 3.0 mg/mouse, administered twice, 2 weeks apart). In addition, aspirin treatment did not depress the in vivo formation of BaP metabolite:DNA adducts in lung or liver at either dose of BaP (6.0 and 0.06 mg/mouse). The lower dose of BaP was used in the adduct study to assess the effect of aspirin at a more environmental exposure level of BaP. Treatment with indomethacin, another PES inhibitor, also did not reduce the pulmonary BaP metabolite:DNA adduct levels. The failure of PES inhibitors to reduce the number of pulmonary adenomas and BaP metabolite:DNA adduct levels suggests that cooxidation of BaP during prostaglandin biosynthesis may not play a significant role in BaP-induced pulmonary adenomas. (Auth. Abs.)

84–1328. Adriaenssens, P. I.; White, C. M.; Anderson, M. W. **Dose-Response Relationships for the Binding of Benzo(*a*)pyrene Metabolites to DNA and Protein in Lung, Liver, and Forestomach of Control and Butylated Hydroxyanisole-Treated Mice.** *Cancer Research* 43(8): 3712-3719, August 1983.

Data on the effect of dietary butylated hydroxyanisole (BHA) on the dose-dependency for benzo(*a*)pyrene (BaP) metabolite:DNA adduct formation in target and nontarget tissues for BaP-induced neoplasia and for binding of BaP metabolites to protein in these tissues are reported. The inhibition of (+)-7β, 8α-dihydroxy-9α, 10α-epoxy-7,8,9,10-tetrahydrobenzo(*a*)pyrene (BPDEI):DNA adduct formation (the major adduct identified in lung, liver, and forestomach of rats), was dose-dependent in lung and liver. Inhibition of lung (68 percent) and liver (82 percent) adduct formation was highest at a BaP dose of 270 μmol/kg. As the BaP dose approached zero, inhibition of BPDEI:DNA adduct formation by BHA decreased with BaP dose and approached values of approximately 40 percent (lung) and 55 percent (liver). The dose-dependency of the binding of BaP metabolites to protein was also examined. BPDEI:DNA adduct concentrations ranged from 2 to 10 percent of protein binding concentrations in liver of untreated animals, from 3 to 7 percent in forestomach, and from 5 to 7 percent in lung. The dose—response curves for protein binding of BaP metabolites in lung and liver from BHA-treated animals were essentially parallel to those in control animals; thus, the inhibition of protein binding by BHA treatment was not dose-dependent in these organs. No consistent BHA effect was observed on the amount of binding of BaP metabolites to forestomach protein. (Auth. Abs. Mod.)

84–1329. Asmussen, I. **Mitochondrial Proliferation in Endothelium. Observations on Umbilical Arteries From Newborn Children of Smoking Mothers.** *Atherosclerosis* 50(2): 203-208, February 1984.

During analysis of umbilical arteries from 22 heavy smokers (>10 cigarettes/day) and 30 nonsmokers, studied by transmission electron microscopy, an increased number of mitochondria was observed in many endothelial cells of smokers ($\chi^2 = 16.49$). The mean number of mitochondrial profiles was 23/endothelial cell, compared with 3/cell in nonsmokers. Mitochondrial volume density (area covered by mitochondrial profiles/area cytoplasm) was measured and found to range from 0.15 to 0.62 in smokers compared with 0.01 to 0.02 in nonsmokers. Mean mitochondrial index (mitochondrial volume density/number of mitochondrial profiles) was 0.018 in smokers and 0.006 in nonsmokers. Thus, in smokers a larger area of the endothelial cell cytoplasm was often occupied by mitochondria, and a greater number of mitochondrial profiles was found, but the mean size of the mitochondria (profiles) was only slightly increased. The findings have implications for recent research on endothelial metabolic capacity and its changes in relation to atherosclerosis. (Auth. Abs. Mod.)

84–1330. Backon, J. **Relative Female Superiority in Resistance to Malignant Transformation Induced by Radiation and Smoking: Due to Higher Basal Prostaglandin Levels and Lower Essential Fatty Acid Needs? (Letter).** *Biomedicine and Pharmacotherapy* 37(8): 405, 1983.

The recent observation (*Biomedicine and Pharmacotherapy* 36(6/7): 275-276, 1982) that women may be more resistant than men to malignant transformation induced by radiation or smoking may be related to the relative need for essential fatty acids and, consequently, basal prostaglandin synthesis. Prostaglandin E_1 (PGE_1) seems to be required for, and enhance the activity of, thromboxane (TXA_2), derived from arachidonic acid, since it is able to activate TXA_2 receptors. Oxygenated hemoglobin may be important in the formation of TXA_2 and thus may be reduced in smokers. Transformed cells in culture can be made to undergo reverse transformation and be normalized by exposure to cyclic AMP, PGE$_1$, and TXA$_2$. If the basal rate of essential fatty acids need is less in females than in males, it is possible that enhanced PGE$_1$ synthesis is more effective in males than in females. See also 84-1397.

84–1331. Balconi, G.; Pietra, A.; Busacca, M.; De Gaetano, G.; Dejana, E. **Success Rate of Primary Human Endothelial Cell Culture From Umbilical Cords Is Influenced by Maternal and Fetal Factors and Interval From Delivery.** *In Vitro* 19(11): 807-810, November 1983.

Successful in vitro cultivation of endothelial cells from human umbilical cord vein was impaired by maternal smoking and by use of cords ≤ 1 hour after delivery. Of the 109 cords analyzed, 22 were from women who had smoked ≥ 5 cigarettes/day, 11 of whom had consumed >15/day. Smoking by the mother negatively influenced the rate of culture success, especially among heavy smokers. Only three cultures were obtained from the cords of women smoking >15 cigarettes/day. The mechanisms leading to smaller numbers of cells in cord specimens processed shortly after delivery (15 percent successful cultures) remain to be established, since specimens processed 5 to 24 hours after delivery produced successful cultures 70 to 100 percent of the time. Age, parity, diabetes, and hypertension in the mother, type of delivery, and sex and weight of the newborn did not significantly influence successful establishment of endothelial cell cultures. (Auth. Abs. Mod.)

84–1332. Benedek, I. H.; Blouin, R. A.; McNamara, P. J. **Influence of Smoking on Serum Protein Composition and the Protein Binding of Drugs.** *Journal of Pharmacy and Pharmacology* 36(3): 214-216, March 1984.

Smokers had significantly higher serum α_1-acid glycoprotein (α_1-AGP) levels compared with nonsmokers in an investigation of smoking's influence on serum proteins and the protein binding of phenytoin and propranolol. Mean α_1-AGP concentrations were 84.3 mg/dl in smokers and 62.8 mg/dl in nonsmokers ($p<0.05$). A trend for lower serum albumin concentrations and lower fraction unbound of propranolol among smokers was found, but smoking did not affect the protein binding of phenytoin. (Auth. Abs. Mod.)

84–1333. Benowitz, N. L.; Jacob, P., III. **Daily Intake of Nicotine During Cigarette Smoking.** *Clinical Pharmacology and Therapeutics* 35(4): 499-504, April 1984.

Daily intake of nicotine in 22 subjects was estimated from metabolic clearance data obtained after intravenous infusion of nicotine, and from blood and urinary nicotine concentrations over 24 hours when subjects were smoking cigarettes. Daily intake of nicotine averaged 37.6 mg (± 17.7, SD), but varied widely among subjects (10.5 to 78.6 mg). Men metabolized nicotine faster than women, but daily intake of nicotine did not differ. Intake correlated strongly with cigarettes/day ($r=0.59$) but not with machine-determined yield. Nicotine intake/cigarette averaged 1.04 mg (± 0.36), but did not correlate with machine-determined yield. Correlations between several commonly used biochemical markers of tobacco smoke and nicotine intake were examined, and the afternoon (4:00 p.m.) blood level of nicotine was found to be the best marker. (Auth. Abs.)

84–1334. Boksa, P.; Livett, B. G. **Substance P Protects Against Desensitization of the Nicotinic Response in Isolated Adrenal Chromaffin Cells.** *Journal of Neurochemistry* 42(3): 618-627, March 1984.

Substance P (10^{-5} *M*), a peptide endogenous to the splanchnic nerve, completely protected against desensitization of catecholamine release produced by acetylcholine at 37° C or 23° C and by nicotine at 23° C and afforded appreciable protection against nicotine-induced desensitization at 37° C. The peptide had no effect on K^+-induced desensitization of catecholamine release. Like substance P, *d*-tubocurarine also prevented nicotinic desensitization. Substance P prevented two components of nicotinic desensitization, i.e., the Ca^{2+}-dependent component and the Ca^{2+}-independent, depletion-independent component. Substance P had little effect on subsequent catecholamine uptake, indicating that the protection against desensitization is a result of facilitation of catecholamine release rather than inhibition of catecholamine reuptake. Nicotine-induced catecholamine release and nicotinic desensitization of catecholamine release were Na^+-independent, although substance P's inhibition of nicotine-induced catecholamine release was reduced by extracellular Na^+. Substance P may play a similar role in vivo. Its protection against nicotinic desensitization may ensure a maintained output of adrenal catecholamines during stress, when the splanchnic nerve releases large amounts of acetylcholine. (Auth. Abs. Mod.)

84–1335. Boksa, P.; Livett, B. G. **Desensitization to Nicotinic Cholinergic Agonists and K^+, Agents That Stimulate Catecholamine Secretion, in Isolated Adrenal Chromaffin Cells.** *Journal of Neurochemistry* 42(3): 607-617, March 1984.

Prior exposure of cultured bovine adrenal chromaffin cells to the nicotinic cholinergic agonists acetylcholine (ACh) or nicotine caused a subsequent depression or desensitization of catecholamine (CA) release during restimulation with the same agonists. Rates of development of and recovery from nicotinic desensitization were in the minute time range and the magnitude of nicotinic desensitization of CA release was greater at 37°C than at 23°C. ACh- or nicotine-induced desensitization was shown to be the result of two processes: (1) a Ca^{2+}-dependent component of desensitization, possibly due to a depletion of intracellular CA stores; and (2) a Ca^{2+}-independent, depletion-independent component of desensitization. Prior exposure of cultured chromaffin cells to an elevated concentration of K^+ also resulted in desensitization of K^+-induced CA release. K^+-induced desensitization was completely Ca^{2+}-dependent and was shown to be the result, at least in part, of a mechanism that is independent of depletion of CA stores. (Auth. Abs. Mod.)

84–1336. Cannon, D. J.; Casteel, L.; Read, R. C. **Abdominal Aortic Aneurysm, Leriche's Syndrome, Inguinal Herniation, and Smoking.** *Archives of Surgery* 119(4): 387-389, April 1984.

Cigarette smoking has been identified as an important risk factor for abdominal aortic aneurysm (AAA) and Leriche's syndrome (LS). Earlier studies demonstrated an increase in serum proteolytic activity in male smokers with direct inguinal herniation and a similar imbalance in smokers with AAA, but not in smokers with LS. To determine whether the protease imbalance in the blood of smokers with AAA or herniation is a causal factor, 417 LS patients served as controls. Frequency of inguinal herniation was significantly higher in 341 AAA patients (25.8 percent) compared with LS patients (14.6 percent). Patients with AAA also had more severe herniation (direct, bilateral, recurrent, or earlier onset) and had more pronounced leukocytosis (9000/cu mm vs. 8190/cu mm). Data suggest that increased blood proteolytic activity may play a role in the development of both AAA and adult inguinal herniation, but not LS. Men who smoke manifest different systemic effects.

84–1337. Castonguay, A.; Tjalve, H.; Trushin, N.; Hecht, S. S. **Perinatal Metabolism of the Tobacco-Specific Carcinogen 4-(Methylnitrosamino)-1-(3-pyridyl)-1-butanone in C57BL Mice.** *Journal of the National Cancer Institute* 72(5): 1117-1124, May 1984.

Whole-body autoradiography of pregnant C57BL mice given i.v. injections of carbonyl-^{14}C-labeled 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) revealed that some metabolites were covalently bound to cellular macromolecules of the nasal mucosa, bronchial mucosa, and liver of the mother. Unbound metabolites were present in those tissues and were also localized in the gastrointestinal lumens, kidneys, urinary bladder, eye melanin, and corpora lutea of the ovaries. Autoradiograms taken at the 1-hour survival interval showed labeling of the fetal kidneys and urinary bladder. At the 4-hour survival interval, amniotic fluid was strongly labeled. Chromatography of extracts of tissues obtained from pregnant mice on days 13 and 18 of gestation indicated that unchanged NNK and 4-(methylnitrosamino)-1-(3-pyridyl)butan-1-ol (NNAl) were present in placentas and fetal tissues. Some NNK metabolites were covalently bound to tissues of the nose, lung, and liver of 18-day-old fetuses. Results indicate that NNK and NNAl have the capacity to cross the placental barrier and to be activated in fetal tissues. Nasal, pulmonary, and hepatic tissues of 16- and 18-day-old fetuses incubated in vitro with NNK metabolized NNK and NNAl by α-carbon hydroxylation. These pathways lead to intermediates able to alkylate cellular macromolecules. Nasal tissues had a higher capacity to α-carbon hydroxylate NNK than pulmonary or hepatic tissues during the last stage of fetal development and during all stages of postnatal life. Results suggest that exposure of C57BL mice to NNK during the last stage of fetal development or during neonatal life could possibly result in development of tumors. (Auth. Abs.)

84–1338. Cena, V.; Nicolas, G. P.; Sanchez-Garcia, P.; Kirpekar, S. M.; Garcia, A. G. **Pharmacological Dissection of Receptor-Associated and Voltage-Sensitive Ionic Channels Involved in Catecholamine Release.** *Neuroscience* 10(4): 1455-1462, 1983.

Data from comparisons of nicotinic cholinoceptor channels and voltage-sensitive channels during the evoked release of (^{3}H)noradrenaline from cultured bovine adrenal chromaffin cells indicated that (1) imipramine and cocaine inhibited (^{3}H)noradrenaline release by selective inactivation of the acetylcholine receptor ionophore; (2) Na^{+} deprivation blocked nicotine-evoked release; (3) tetrodotoxin had no effect on amine release evoked by receptor activation; and (4) nitrendipine (a potent Ca^{2+} antagonist) equally inhibited nicotine- and K^{+}-evoked release. Based on findings, a mechanism for catecholamine release in stress situations is postulated.

84–1339. Chang, W.-C.; Fukuda, S.; Tai, H.-H. **Pulmonary NAD^{+}-Linked 15-Hydroxyprostaglandin Dehydrogenase Activity Is Decreased by Cigarette Smoking.** *Life Sciences* 34(13): 1261-1268, March 26, 1984.

NAD^{+}-dependent 15-hydroxyprostaglandin dehydrogenase activity, the key enzyme responsible for biological inactivation of prostaglandins, was found to decrease in the lung, but not in the kidney or stomach, following cigarette smoke exposure. To determine the possible effect of cigarette smoking on vascular homeostasis, male rats were exposed to freshly generated cigarette smoke once daily for 4 to 13 weeks. Inhalation of smoke was verified by elevated level of carboxyhemoglobin. The consequence of impaired pulmonary metabolism of prostaglandins and thromboxane may result in alteration of vascular homeostasis and subsequently lead to cardiovascular disorders commonly found in smokers. (Auth. Abs. Mod.)

84–1340. D'Arcy, P. F. **Drug Interactions and Reactions Update. Tobacco Smoking and Drugs: A Clinically Important Interaction?** *Drug Intelligence and Clinical Pharmacy* 18(4): 302-307, April 1984.

There is little evidence, in the majority of examples, that smoking and drug interactions are a recognizable hazard. Exceptions are limited at present to four drugs: insulin, propoxyphene, propranolol, and theophylline preparations, where there is some clinical evidence of an interaction with tobacco smoking. It has been demonstrated that smoking may interact with the metabolism of a number of drugs, but smoking as a variant has been studied with relatively few clinically essential drugs. This review records data from pharmacokinetic or other studies and attempts to determine whether such interactions have clinical significance. Ten drugs that are known to be affected by smoking were investigated. The recognized association between smoking and the use of oral contraceptives by women is not discussed, since it is a specialized problem. It is suggested that more studies are needed on more drugs. (Auth. Abs. Mod.)

84–1341. Emura, M.; Mohr, U. **Toxic and Transforming Effects of Cigarette Smoke Condensates in Fetal Syrian Golden Hamster Lung Cell Cultures.** *Experimental Pathology* 24(4): 261-264, 1983.

Morphological transformation of cultured mammalian cells may provide a possible predictive system for tumorigenicity and co-tumorigenicity of carcinogens such as those found in cigarette smoke. The cytotoxic and transforming effects of cigarette smoke condensates (CSCs) and benzo(*a*)pyrene (BaP) were compared using cultures of fetal Syrian golden hamster lung cells. The dose—response patterns differed completely between CSCs and BaP, and the amount of BaP alone did not explain the transforming activity of CSCs. It is suggested that impure substances such as CSCs show dose—response patterns that differ greatly from those of any purified, single ingredient, owing to suppressive or potentiating effects caused by multiple interactions among the numerous condensate constituents.

84–1342. Fortmann, S. P.; Rogers, T.; Vranizan, K.; Haskell, W. L.; Solomon, D. S.; Farquhar, J. W. **Indirect Measures of Cigarette Use: Expired-Air Carbon Monoxide Versus Plasma Thiocyanate.** *Preventive Medicine* 13(1): 127-135, January 1984.

Two biochemical markers of cigarette use, expired-air carbon monoxide (CO) and plasma thiocyanate (SCN), were significantly higher in smokers compared with nonsmokers in a representative population sample of 2,237 adults (aged 18 to 74) from the baseline survey of the Stanford Five City Project. CO and SCN levels also correlated well with number of cigarettes smoked/day. CO appears to be more sensitive and specific, and misclassifies significantly fewer nonsmokers, regular smokers, and light smokers (less than nine cigarettes/day) than SCN. A combination of CO and SCN classifies smokers and nonsmokers better than either does alone. Neither marker is a reliable indicator in irregular smokers (no cigarettes in the past 48 hours). Despite a much shorter metabolic half-life, CO was a better indicator of cigarette use than SCN in this cross-sectional sample. CO is generally simpler and less expensive to measure than SCN, and may be a preferable indirect measure of smoking status in studies of smoking cessation. (Auth. Abs. Mod.)

84–1343. Fraser, H. S.; Dotson, O. Y.; Howard, L.; Grell, G.A.C.; Knight, F. **Drug Metabolizing Capacity in Jamaican Cigarette and Marijuana Smokers and Non-Smokers.** *West Indian Medical Journal* 32(4): 207-211, December 1983.

Antipyrine half-life was 16.4±8.5 hours in nonsmokers, 10.3 hours in tobacco smokers, 10.2 hours in marihuana smokers, and 8.6±1.8 hours in smokers of both cigarettes and marihuana, when antipyrine elimination was studied in 31 black Jamaican men (10 nonsmokers, 7 cigarette smokers, 5 marihuana smokers, and 9 smokers of both marihuana and cigarettes). Use of both marihuana and tobacco was associated with shorter antipyrine half-lives and faster clearance, implying faster metabolism. The order of magnitude was similar, and use of both drugs had an additive effect. It is concluded that smoking habits should be considered when choosing dose regimens for oxidized drugs. (Auth. Abs. Mod.)

84–1344. Ghosh, R.; Ghosh, P. K. **Sister Chromatid Exchanges in Betel and Tobacco Chewers.** *Mutation Research* 139(2): 79-81, February 1984.

Betel chewers (n=18) and betel-plus-tobacco chewers (n=15) showed higher sister chromatid exchanges (SCE) compared with controls (n=20). Betel chewing, a widespread habit in India, is practiced in a variety of ways, with quid that varies in composition in different parts of the country. The 53 male nonsmokers studied were not occupationally exposed to other mutagens, and included the 20 apparently normal nonchewers. The incidence of SCE was investigated in lymphocyte chromosomes, and differences were statistically evaluated using the paired t-test. Higher SCE frequencies were observed in chewers with higher daily consumption and in those who had practiced the habit for >10 years.

84–1345. Hara, E.; Kawajiri, K.; Tagashira, Y. **Immunochemical Study on the Contributions of Two Molecular Species of Microsomal Cytochrome P-450 to the Metabolism of Benzo(*a*)pyrene by Rat Liver Microsomes.** *Cancer Research* 43(8): 3604-3608, August 1983.

The roles of two species of cytochrome P-450, the major cytochrome P-450 components of liver microsomes of phenobarbital-treated rats (PB-P-450) and 3-methylcholanthrene-treated rats (MC-P-448), were studied in the metabolism of benzo(*a*)pyrene (BaP) in rat liver microsomes in vitro. BaP was incubated with polychlorinated biphenyl-treated rat liver microsomes, in which PB-P-450 and MC-P-448 constituted about 45 and 24 percent of the total cytochrome P-450, respectively. The metabolites were then separated into those soluble in ethyl acetate and in water and those covalently bound to protein. Using high pressure liquid chromatography, the ethyl acetate-soluble metabolites were separated into three major groups, phenols, quinones, and dihydrodiols, and three unknown materials. Addition of anti-MC-P-448 immunoglobulin to the reaction mixture completely inhibited the formation of all ethyl acetate-soluble metabolites. In contrast, anti-PB-P-450 immunoglobulin did not inhibit the formation of 4,5-dihydro-4,5-dihydroxybenzo(*a*)pyrene and 3-hydroxybenzo(*a*)pyrene; partially inhibited the formation of 7,8-dihydro-7,8-dihydroxybenzo(*a*)pyrene, 9,10-dihydro-9,10-dihydroxybenzo(*a*)pyrene, and the three unknown materials; and caused 30 to 40 percent enhancement of the formation of 9-hydroxy-benzo(*a*)pyrene and benzo(*a*)pyrene-3,6-dione and 80 percent enhancement of benzo(*a*)pyrene-1,6-dione. Antibody against MC-P-448, but not against PB-P-450, also caused 75 percent inhibition of the formation of water-soluble metabolites and 85 percent inhibition of formation of protein-bound BaP metabolites. Results show that MC-P-448 is important in the metabolism of BaP. (Auth. Abs.)

84–1346. Hirsch, J.-M.; Svennerholm, B.; Vahlne, A. **Inhibition of Herpes Simplex Virus Replication by Tobacco Extracts.** *Cancer Research* 44(5): 1991-1997, May 1984.

Extracts of snuff have inhibitory effects on the production of cytolytic *Herpes simplex* virus type 1 (HSV-1) infections. Plaque formation assays of HSV-1 in the presence of snuff extract showed a reduced number of plaques. Different batches of one brand of snuff were tested for inhibition of *Herpes simplex* virus (HSV) production. More than 99 percent inhibition of 24-hr HSV production was obtained with undiluted batches. The 1 to 5 dilutions of snuff had an inhibitory effect of 85 percent and 1 to 25 dilutions, 39 percent. Attachment of the virus to the host cell, penetration of the virus to the cell nuclei, and synthesis of viral DNA were inhibited. Nicotine had an inhibitory effect, while aromatic additions to snuff were found to have no major inhibitory effect on HSV replication. Brands with reported high levels of tobacco-specific *N*-nitrosamines had significantly greater ability to inhibit HSV replication. Because a chronic snuff dipper keeps tobacco in the mouth for most of the day, it is suggested that an interaction between tobacco products and HSV-1 might be involved in development of dysplastic lesions in the oral cavity. (Auth. Abs. Mod.)

84–1347. Hogstedt, B. **Micronuclei in Lymphocytes With Preserved Cytoplasm: A Method for Assessment of Cytogenetic Damage in Man.** *Mutation Research* 130(1): 63-72, February 1984.

Multivariate analysis revealed a significant effect for smoking (p=0.014) and for low-dose styrene exposure (p=0.005) on micronuclei in lymphocytes with preserved cytoplasm in 96-hour cultures in a comparison of methods for analyzing cytogenetic damage. Cells from 58 subjects (38 styrene exposed) were analyzed after (1) hypotonic treatment or (2) cytoplasm preservation. Maximum frequency of micronuclei was obtained in 80 to 88 hour cultures in cells X-rayed in vitro and in 96 hours in nonirradiated cells. Whole blood and buffy coat preparations correlated closely, as did two observers' findings, but a systematic difference of about 30 percent existed between the two sets of observations. Method error (variation coefficient) was 11 and 6 percent in preparations with mean micronuclei frequencies of 4 and 38 percent, respectively. The styrene-exposed group displayed weak but statistically significant correlations between frequencies of micronuclei and numerical chromosome aberrations. Smoking, styrene exposure, and age explained only 12 to 24 percent of the total variance in micronuclei. It is noted that smokers can be used as positive controls in cytogenetic studies.

84–1348. Hopkin, J. M.; Evans, H. J. **Cellular Effects of Smoke From "Safer" Cigarettes.** *British Journal of Cancer* 49(3): 333-336, March 1984.

On a weight-for-weight basis, cigarette smoke condensates (CSC) from low-, middle-, and high-tar cigarettes produce similar mutagenic effects (detected by induced sister chromatid exchanges), and similar cytotoxic effects (determined by vital dye exclusion) in human leukocytes. CSC from a variety of cigarettes available on the British market were collected on automatic smoking apparatus and assessed for mutagenicity and cytotoxicity. Although filters reduced the smoke yield of cigarettes, on an equal weight basis the CSC from different tar and filter cigarette categories showed similar cytotoxic and mutagenic properties. These findings, in addition to strong evidence that smokers extract more smoke from lower tar cigarettes to compensate for low nicotine yields, suggest that health dangers associated with smoking "safer" products are underestimated. (Auth. Abs. Mod.)

84–1349. Kaelin, A. C.; Cummings, A. J. **Differential Inhibition of Human Placental Monooxygenase Activity: Evidence for Multiple Forms of 7-Ethoxycoumarin O-Deethylase.** *Biochemical Pharmacology* 33(3): 505-508, February 1, 1984.

Evidence is provided for the existence of at least two different forms of 7-ethoxycoumarin *O*-deethylase activity in the human placenta; one form, induced by maternal smoking, was strongly inhibited by *a*-naphthoflavone, moderately inhibited by metyrapone, and weakly inhibited by dimethylsulfoxide (DMSO). The other form, present in placentas from nonsmokers, was strongly inhibited by DMSO and metyrapone, but weakly by *a*-naphthoflavone. Several parallels between the human placental monooxygenase system and that of rat liver are described. It is concluded that the deethylase activity in nonsmokers' placentas is associated with cytochrome P-450 and is qualitatively different from the activity induced by maternal smoking, which appears to be associated with cytochrome P-448.

84–1350. Lichtensteiger, W.; Schlumpf, M.; Davis, M. D. **Catecholamines and Nicotine in Early Neuroendocrine Organization.** In: Cohen, M. M.; Schlumpf, M.; Lichtensteiger, W. (Editors). *Drugs and Hormones in Brain Development.* Monographs in Neural Sciences, Volume 9. Basel, Karger, 1983, pp. 213-224.

The functional state of adult neuroendocrine transmitter systems is affected by prenatal exposures to nicotine and other factors. Nicotine affects many hormone axes of the adult organism, and chronic administration during pregnancy results in behavioral disturbances in the offspring. For instance, the sex difference in saccharin preference that is distinct in control rats is completely lost in the offspring of nicotine-treated rats, suggesting that prolonged neonatal nicotine exposure can interfere with the sexually dimorphic organization of the brain. Conceivably, this is linked with alterations in fetal testosterone levels. An investigation of serum testosterone in male rat fetuses showed nicotine (25 μg/100 g/hr) suppressed the peak at gestational day 18, without shifting the peak to another day (as in the case of exposure to maternal stress). The early organization of pituitary control by the tubero-hypophyseal dopamine system, with respect to intermediate lobe function, is also discussed.

84–1351. Lilis, R.; Valciukas, J. A.; Weber, J.-P.; Malkin, J.; Selikoff, I. J. **Epidemiologic Study of Renal Function in Copper Smelter Workers.** *Environmental Health Perspectives* 54: 181-192, March 1984.

Cigarette smoking contributed significantly to blood cadmium (Cd-B) levels, but not to blood lead (Pb-B) levels in 920 copper smelter employees. Subjects included active smelter employees, retired workers, and copper mine employees who had never worked in the smelter. Slight to moderate absorption of lead and cadmium was definitely present in active copper smelter employees, who had significantly higher levels of Pb-B, Cd-B, and blood zinc protoporphyrin (ZPP) than retired employees and miners. A marked effect of smoking on Cd-B was present, but for all smoking categories, levels were higher in active employees, indicating an independent contribution. Median Cd-B level for active workers was 2.75 μg/L (range 6.93±4.10 in smokers to 1.04±0.96 in never smokers). Urinary cadmium (Cd-U) levels were higher in retired workers, who were also older and had longer duration of exposure in the smelter. Cd-U did not exceed 10 μg/g creatinine (the level considered critical for nephrotoxicity) in any subject. A relatively small proportion (16.7 percent) of active employees showed Pb-B levels of 40 μg/dL or higher. Although smokers in all categories had slightly higher Pb-B levels, the effect was not significant. There were no significant correlations between urinary β_2-microglobulin levels and Cd-U, Cd-B, Pb-B, and ZPP or between urinary β_2-microglobulin excretion and serum creatinine or blood urea nitrogen levels. That kidney function could be impaired by long-term exposure in the smelter was only indirectly suggested. Effects on renal function at the low levels of cadmium and lead absorption that were observed in this smelter population were minimal. (Auth. Abs. Mod.)

84–1352. Littlewood, J.; Glover, V.; Sandler, M.; Peatfield, R.; Petty, R.; Rose, F. C. **Low Platelet Monoamine Oxidase Activity in Headache: No Correlation With Phenolsulphotransferase, Succinate Dehydrogenase, Platelet Preparation Method or Smoking.** *Journal of Neurology, Neurosurgery, and Psychiatry* 47(4): 338-343, April 1984.

Analysis of male migraine (n=19) and cluster headache (n=11) patients showed that relationships of platelet monoamine oxidase (MAO) and smoking to headache syndromes are independent of each other. Platelet MAO activity was lower in all headache patients, while incidence of smoking was higher in cluster headache patients (95 percent) compared with migrainous group patients (26 percent) or 11 controls (23 percent). Low platelet MAO activity was observed in nonsmokers, and lower activity was seen in smoking patients compared with smoking controls. Also, in five subjects who quit smoking during the study, platelet MAO activity remained constant through 3 months of followup. Use of Corash's platelet preparation method showed low activity values were not due to abnormal recovery. Although low succinate dehydrogenase activity was identified in headache subjects, it was a separate phenomenon and no correlation was found between the activity of MAO and phenolsulfotransferase M or succinate dehydrogenase.

84–1353. Maitra, S. C.; Chakraverty, K.; Shipstone, A. C.; Kar, K. **Ultrastructural Changes in the Neural Lobe of the Rat Pituitary Following Nicotine Pretreatment.** *Experimental and Clinical Endocrinology* 82(3): 376-379, 1983.

The effect of nicotine on ultrastructural changes and hormone contents of the neural lobe of the pituitary were studied in 14 male albino rats. Nicotine caused a significant release of both vasopressin and oxytocin from the neural lobe. Examination of the neural lobe with electron microscopy revealed the nerve terminals were abruptly depleted of neurosecretory granules. Loss of electron dense material occurred within 10 min of injection of nicotine. (Auth. Abs. Mod.)

84–1354. Malonova, H.; Bardodej, Z. **Urinary Excretion of Mercapturates as a Biological Indicator of Exposure to Electrophilic Agents.** *Journal of Hygiene, Epidemiology, Microbiology, and Immunology* 27(3): 319-328, 1983.

An increase in the urinary excretion of mercapturates was found in individuals who smoked and in those exposed to styrene, aromatic hydrocarbons, dimethylformamide, and 1,1,1-trichloroethane. Excretion of mercapturates was analyzed photometrically in individuals exposed to styrene, a mixture of aromatic hydrocarbons, butadiene, vinyl chloride, 1,1,1-trichloroethane, trichloroethylene, tetrachloroethylene, 1,1,1-trifluoro-2-bromo-2-chloroethane (Halothane), ethylene oxide, epichlorhydrin, bis(chloromethyl)-ether, N-methylacrylamide, dimethylformamide, nitrosamines, and cis-platinum. Subjects were divided according to smoking status, sex, and rural vs. urban residence. No sex-related differences were observed. The excretion rate in unexposed smokers was significantly higher than that in nonsmokers, with the highest rates among smokers residing in an industrial town. The lowest mercapturate concentrations were found among nonsmoking controls from a mountain village where air pollution due to motor vehicle emissions was lowest. Lack of elevated mercapturate excretion in those exposed to other industrial chemicals is considered an indication of careful handling and, thus, low exposure levels. (Auth. Abs. Mod.)

84–1355. McSharry, C.; Banham, S. W.; Lynch, P. P.; Boyd, G. **Antibody Measurement in Extrinsic Allergic Alveolitis.** *European Journal of Respiratory Diseases* 65(4): 259-265, May 1984.

Cigarette smoking impaired antibody production among the active pigeon fanciers studied; 27 of 145 smokers were antibody responders compared with 111 of 220 nonsmokers who were antibody positive, despite similar antigen exposure in both groups. Mean immunoglobulin G (IgG) level of antibody-positive smokers was also lower than among nonsmokers, 17.1 and 43.7 μg/ml, respectively. Serum IgG antibody against purified avian antigens was quantified by radioimmunoassay in 507 pigeon fanciers, of whom 110 had documented pigeon breeders disease (PBD). The incidence of PBD increased from 3.7 percent in the antibody-negative group to 78 percent of those with IgG antibody greater than 30 μg/ml. The highest levels of antibody tended to occur in subjects with relatively short avian exposure, and a significant correlation between decreasing antibody levels and increasing years of avian contact was demonstrated that was unrelated to age or smoking history. The antibody-positive group (which contained most of the PBD subjects) had lower daily hours of avian contact and demonstrated some self-regulation of antigen exposure. (Auth. Abs. Mod.)

84–1356. Niederman, M. S.; Fritts, L. L.; Merrill, W. W.; Fick, R. B.; Matthay, R. A.; Reynolds, H. Y.; Gee, J.B.L. **Demonstration of a Free Elastolytic Metalloenzyme in Human Lung Lavage Fluid and Its Relationship to Alpha$_1$-Antiprotease.** *American Review of Respiratory Disease* 129(6): 943-947, June 1984.

Mean elastolytic activity in concentrated human lung lavage fluid (HLF) of smokers ($n=15$) was 18.9 ± 6.7, significantly higher than in nonsmokers ($n=10$, mean activity 4.4 ± 1.8). Activity was measured against ^{14}C elastin substrate and expressed as ng porcine pancreatic elastase equivalents/mg lavage fluid protein. With the synthetic elastin-like chromophore substrate succinyl-trialanine-nitroanilide, activity in individual samples was reduced 79 percent by EDTA, a metalloproteinase inhibitor, but by only 29 percent in the presence of PMSF, a serine proteinase inhibitor. With a pooled HLF sample and ^{14}C elastin substrate, 80 percent of activity was eliminated by EDTA and 51 percent by PMSF. Activity measured with ^{14}C elastin substrate correlated inversely with antigenic α_1-antiprotease ($r=-0.5$, $p=0.01$), but no correlations were found between activity and smoking history, HLF cell number, cell viability, or differential count. Detection of activity with ^{14}C elastin in HLF, with primarily a metalloenzyme inhibitor profile, in the presence of antigenically detectable α_1-antiprotease, may have pathogenetic relevance for emphysema in humans. (Auth. Abs. Mod.)

84–1357. Pasternak, G.; Schlott, B.; Gryschek, G.; Albrecht, S.; Reinhofer, J.; Matthes, E.; Von Broen, B. **Lymphocyte Reactivity in Normal and Malignant Cell Proliferation Against a Phylogenetically Conserved Antigen in Fetal Extracts.** *Journal of the National Cancer Institute* 72(4): 901-908, April 1984.

Injection of benzo(*a*)pyrene (BaP) into mice produced lymphocyte reactivity to fetal extract, with the dose inversely related to the latency of the appearance of reactivity. Lymphocyte reactivity to extracts from fetuses of different species was measured by the macrophage electrophoretic mobility (MEMB) and/or the leukocyte migration inhibition tests in tumor-bearing mice and humans. In chemical carcinogenesis, reactivity was detectable before tumors developed. Six weeks after s.c. injection of 1,000 μg BaP into XVII/Bln mice, the MEMB test became positive. The latent period was 15 weeks after 1.0 μg BaP. Painting of mouse skin with 7,12-dimethylbenz(*a*)anthracene (DMBA), croton oil, or benzene had the same sensitizing effect as BaP. In contrast to the strong carcinogens BaP and DMBA that caused lymphocyte reactivity to persist until tumors developed, benzene and the promoter croton oil induced only a transient effect. Termination of treatment abolished reactivity within 12 weeks. Lymphocyte reactivity to fetal extract in normal cell proliferation was evident since two-thirds-hepatectomized rats and BCG-treated mice became MEMB-positive. In hepatectomized rats the effect was reversible according to the completion of liver regeneration. (Auth. Abs. Mod.)

84–1358. Riebe, M.; Westphal, K. **Studies on the Induction of Sister-Chromatid Exchanges in Chinese Hamster Ovary Cells by Various Tobacco Alkaloids.** *Mutation Research* 124(3/4): 281-286, December 1983.

Since conflicting results have been published on the effect of nicotine on genetic material, the ability of tobacco alkaloids to induce chromosomal aberrations or sister chromatid exchanges (SCEs) in eucaryotic cells was investigated in Chinese hamster ovary cells. Of five tobacco alkaloids tested, only anatabine led to a dose-dependent enhancement of the number of SCEs. Nicotine and nornicotine at high concentrations increased the baseline frequency of SCEs, while anabasine and myosmine had no influence on spontaneous SCE frequency. Results imply that compounds other than tobacco alkaloids are probably responsible for the mutagenic activity noted in cigarette smoke condensate. Since cigarette smoke condensate is a mixture of from 5,000 to 10,000 substances, it is impossible to identify one or more individual substances as being responsible for this activity. (Auth. Abs. Mod.)

84–1359. Ruchirawat, M.; Saengchan, S. **Effects of Riboflavin on the Dimethylnitrosamine Demethylase System in the Rat Liver.** *Journal of the National Cancer Institute* 72(5): 1167-1171, May 1984.

The effects of riboflavin on the dimethylnitrosamine ((DMN), *N*-nitrosodimethylamine) demethylase system in inbred F344 rat liver were investigated. Dietary riboflavin deficiency markedly stimulated the activity of DMN demethylase I operating at a low substrate concentration (4 mM), but caused no change in the activity of DMN demethylase II operating at a high substrate concentration (200 mM). This effect could be reversed by short-term supplementation of the vitamin by a series of three i.p. injections (0.5 mg/dose/day) to the deficient animals. This dosage regimen of riboflavin did not change the activity of DMN demethylase I in the normal animals. In vitro data demonstrated that flavins were inhibitory to DMN demethylase I. Enzyme activity in the microsomes of both control and deficient animals was inhibited by preincubation with flavins, suggesting that the enzyme—flavin interaction rendered the enzyme inactive. Flavin adenine dinucleotide appeared to have a more pronounced effect than riboflavin. (Auth. Abs. Mod.)

84–1360. Scott, R.; Aughey, E.; Reilly, M.; Cunningham, C.; McClelland, A.; Fell, G.S.F. **Renal Cadmium Content in the West of Scotland.** *Urological Research* 11(6): 285-290, 1983.

Significantly higher renal cortex and medulla cadmium levels were found in smokers compared with nonsmokers, and a significant difference in levels ($p<0.05$) between smokers dying from ischemic heart disease and nonsmokers dying from similar causes. Cadmium, zinc, and calcium levels were measured in 290 human kidneys. Cortical vs. medullary levels were found to be significantly different for all three elements. Statistically different levels were also found in those who died of cancer compared with victims of pulmonary disease. Nonsmokers had significantly less cadmium than smokers, but it was in those who smoked between 10 and 25 cigarettes/day that the greatest differences were found.

84–1361. Sharma, R. P.; Kjellstrom, T.; McKenzie, J. M. **Cadmium in Blood and Urine Among Smokers and Non-Smokers With High Cadmium Intake via Food.** *Toxicology* 29(1/2): 163-171, December 1983.

Results showed that cigarette smoking had a more pronounced and significant effect on whole blood cadmium (Cd) levels than intake of Cd through consumption of oysters. In New Zealand, a widely consumed species of oyster contains an average of 5 μg Cd/g wet weight. Cadmium intake and blood and urinary Cd levels in a group of 78 people with a known high oyster consumption were determined and compared with levels in 17 nonsmoking men and women who did not eat oysters (controls). Subjects were interviewed, and information on smoking habits, diet, medical, and occupational history was obtained. Data show that in spite of a very high intake of cadmium via oysters, the concentration of Cd in whole blood and urine was not greatly elevated in proportion to the intake. (Auth. Abs. Mod.)

84–1362. Sorsa, M.; Falck, K.; Heinonen, T.; Vainio, H.; Norppa, H.; Rimpela, M. **Detection of Exposure to Mutagenic Compounds in Low-Tar and Medium-Tar Cigarette Smokers.** *Environmental Research* 33(2): 312-321, April 1984.

Results indicate that even low-tar (LT) cigarettes (approximately 5 mg/cigarette) are able to induce a high level of carboxyhemoglobin in the blood, an increase in excretion of thioethers, and mutagenicity in the urine. LT cigarettes also caused chromosome damage in peripheral blood lymphocytes. Differences between effects of LT and medium-tar (MT) cigarettes (16 mg/cigarette) were insignificant. Volunteers (males aged 21.0±2.7 years), who had recently entered military service, were divided into three groups: 15 smokers of high- or medium-tar cigarettes; 15 who smoked LT cigarettes; and 12 nonsmokers. The number of cigarettes smoked was recorded daily throughout a 51-day study, and smoking effects were measured at the beginning and after 3-week periods of smoking MT or LT cigarettes. Blood carboxyhemoglobin concentration, thioethers excreted into the urine, mutagenic activity in urine, and frequency of sister chromatid exchanges in blood lymphocytes were measured. For all parameters, differences between smokers and nonsmokers were significant. It is concluded that the definition of the term "safer" cigarettes for brands containing 5 to 10 mg tar is not justified, based on present exposure measurements. (Auth. Abs. Mod.)

84–1363. Staessen, J.; Bulpitt, C. J.; Roels, H.; Bernard, A.; Fagard, R.; Joossens, J. V.; Lauwerys, R.; Lijnen, P.; Amery, A. **Urinary Cadmium and Lead Concentrations and Their Relation to Blood Pressure in a Population With Low Exposure.** *British Journal of Industrial Medicine* 41(2): 241-248, May 1984.

The 24-hour urinary excretion of cadmium (U-Cd) and lead (U-Pb) was independent of both blood pressure and smoking in a 4 percent random population sample of a small Belgian town. Both U-Pb and U-Cd increased with age, and were higher in males than in females. Among men, manual workers excreted more U-Cd (12.6 vs. 7.5 nmol/24 hr) but a similar amount of U-Pb (62 vs. 61 nmol/24 hr) compared with office workers. Mean output in nonsmokers vs. smokers, respectively, was 8.7 vs. 9.6 nmol/24 hr for U-Cd and 61 vs. 65 nmol/24 hr for U-Pb in males, and 7.8 vs. 6.5 nmol/24 hr for U-Cd and 37 vs. 41 nmol/24 hr for U-Pb in females. Thus, although there was a trend toward higher levels among smokers, differences were not significant, possibly because 75 percent of those who smoked consumed less than a pack/day. In simple regression analysis, U-Cd was positively correlated with both systolic ($r=+0.30$) and diastolic ($r=+0.38$) blood pressure in women. However, a weak but negative relationship became apparent between systolic pressure and U-Cd in women ($t=-2.21$) and between diastolic pressure and U-Cd in men ($t=-2.04$). In women, urinary beta-2-microglobulin was related to diastolic pressure ($r=-0.44$) and after adjusting for age, to both systolic ($t=-2.75$) and diastolic ($t=-3.07$) pressure. U-Pb and retinol binding protein did not contribute to blood pressure variability in any of the sex-age groups. (Auth. Abs. Mod.)

84–1364. Stavenow, L.; Falke, P.; Berglund, A. **Effects of Different Injurious Stimuli on Cell Death, Proliferation, and Collagen Secretion by Rabbit Aortic Smooth Muscle Cells and**

Human Umbilical Vein Endothelial Cells in Culture. *Medical Biology* 61(4): 214-218, 1983.

Dimethylsulfoxide-soluble particulate matter in cigarette smoke (DSP) caused changes in rabbit aortic smooth muscle cells exposed to hypoxia similar to effects caused by ultraviolet (UV) light, a well-known cytotoxic stimulus. Proliferation and collagen secretion were reduced, and were not reversible when stimulus was removed after 1 hour. DSP was toxic, since there was an increased leakage of (^{3}H)-thymidine from cells. Increasing DSP concentration caused more pronounced effects, while extending incubation time to 3 hours did not aggravate the effects. Endothelial cells were more sensitive to DSP effects than smooth muscle cells. DSP diluted 1:25 compared with the smallest concentration that had significant effects on smooth muscle cells reduced proliferation of endothelial cells. In dilutions of 1:4, almost all of the cells detached. A possible role for DSP in the development of atherosclerosis is discussed. (Auth. Abs. Mod.)

84–1365. Thomassen, M. J.; Demko, C. A.; Winnie, G. B.; Sherman, J. M. **Function of Human Alveolar Macrophages From Normal Volunteers and Cigarette Smokers: Effect of Cystic Fibrosis Serum.** *Journal of Leukocyte Biology* 35(4): 345-355, April 1984.

Results indicate that alveolar macrophages from heavy smokers are less affected by the inhibitory activity present in cystic fibrosis (CF) serum than macrophages from normal volunteers. Morphologic and biochemical characteristics of these macrophages suggest that they may be in an altered state (activated) compared with alveolar macrophages from normal volunteers. Macrophages from heavy smokers behaved like other "activated" populations in the presence of heated serum, suggesting that the complement receptors of cells from heavy smokers were mediating uptake. Inhibition of *Pseudomonas aeruginosa* phagocytosis by CF serum was significantly less on macrophages from smokers ($p<0.01$). Cells from normal volunteers cultured for 10 days were also less affected by CF serum compared with cells cultured for 24 hours from the same individual ($p<0.01$). It is suggested that complement-mediated opsonization was responsible for attachment or ingestion of *P. aeruginosa* in CF serum by macrophages from heavy smokers and 10-day cultures from normal volunteers that were inhibited by heated CF serum. (Auth. Abs. Mod.)

84–1366. Wald, N. J.; Boreham, J.; Bailey, A. **Relative Intakes of Tar, Nicotine, and Carbon Monoxide From Cigarettes of Different Yields.** *Thorax* 39(5): 361-364, May 1984.

Relative intakes of tar, nicotine, and carbon monoxide (CO) were estimated in 2,455 subjects who freely smoked their usual cigarettes. Indexes of inhaling, measured by dividing carboxyhemoglobin level by CO yield, were analyzed separately by yield and by cigarette type. Yield analysis indicated that inhalation was adjusted to achieve similar nicotine and CO (but not tar) intake regardless of cigarette yield. Analysis by type revealed that inhalation was adjusted to achieve similar tar and nicotine yield, but not CO, which was greater in filtered brand smokers. It is concluded that (1) harmful nicotine—CO effects are unlikely to be reduced by smoking low-yield cigarettes, but tar effects are reduced; and (2) CO effects on the cardiovascular system will be greater in filter cigarette smokers compared with smokers of nonfiltered brands, if they are at similar risk for cardiovascular disease.

84–1367. Wald, N. J.; Boreham, J.; Bailey, A.; Ritchie, C.; Haddow, J. E.; Knight, G. **Urinary Cotinine as Marker of Breathing Other People's Tobacco Smoke. (Letter).** *Lancet* 1(8370): 230-231, January 28, 1984.

A dose—response relationship was found between mean urinary cotinine concentrations in nonsmokers and duration of reported passive exposure to tobacco smoke. Subjects were 221 nonsmoking men who completed a questionnaire on recent passive exposure to smoke and provided a urine sample. Cotinine levels were measured by radioimmunoassay and compared with samples from 232 current smokers. Average cotinine levels in nonsmokers increased about 10-fold from the lowest to the highest quintile of exposure. The median level was much lower than in current smokers. It is concluded that urinary cotinine is a sensitive marker for passive smoke exposure and is likely to be specific, since cotinine is the major metabolite of nicotine, derived solely from tobacco. Results have implications for biochemical epidemiological studies of the hazards of passive smoking.

84–1368. Wargovich, M. J.; Goldberg, M. T.; Newmark, H. L.; Bruce, W. R. **Nuclear Aberrations as a Short-Term Test for Genotoxicity to the Colon: Evaluation of Nineteen Agents in Mice.** *Journal of the National Cancer Institute* 71(1): 133-137, July 1983.

Benzo(*a*)pyrene (BaP), not considered an intestinal carcinogen, induced a weak positive dose—response nuclear aberration in mouse colon. The genotoxicity of 16 agents including several hydrazines, nitrosamines, aromatic amines, polycyclic hydrocarbons, and other related compounds and three known inhibitors of carcinogenesis was assessed in the murine colonic nuclear aberration assay. Of the seven agents considered positive for colonic DNA damage, five were large bowel carcinogens. All structural analogs of the intestinal carcinogens that are tumorigenic for other organs, except BaP, were negative in the colonic nuclear aberration assay, as were all noncarcinogens tested. The metabolic inhibitor disulfiram completely inhibited 1,2-dimethylhydrazine-induced colonic nuclear damage, while inhibition was less marked for the antioxidants butylated hydroxyanisole and caffeic acid. The versatility of the assay as an indicator of colonic genotoxicity resulting from carcinogen exposure is discussed. (Auth. Abs. Mod.)

PHARMACOLOGY AND TOXICOLOGY

84–1369. Weerapradist, W.; Boonpuknavig, V. **Effect of Betel Nut on the Buccal Mucosa of the Hamster Cheek Pouch: Comparative Histological Study of Thai and Indian Betel Nut and Di Methyl Benzanthracene (DMBA).** *Journal of the Dental Association of Thailand* 33(2): 53-66, March-April 1983.

No tumors were apparent in histomorphological observations of the hamster buccal pouch following 30 weeks' application of cured tobacco, Thai betel nut, cured tobacco combined with Thai betel nut, and Indian betel nut. Acanthosis, hyperplasia, and degeneration of basal cells, however, were noted in all groups, in addition to degeneration of mesenchymal tissue and edema in the submucosal layer. Epithelial and submucosal changes in each group showed no significant differences compared with the control group, except for basal cell hyperplasia after application of cured tobacco combined with Thai betel nut. Severity of epithelial and submucosal changes in each group did not correlate with time of application. Dimethyl sulfoxide, used as a solvent for each extract, may have played a role in the epithelial and submucosal changes in all groups. Findings suggest that betel nut and cured tobacco (1) contain no carcinogenic material; (2) contain some carcinogenic material, but the dilutions used were too low to cause tumor development; or (3) other factors are required for tumor induction, such as chronic irritation during mastication. (Auth. Abs.)

See also **84–1377, 84–1393, 84–1452, 84–1500**

MORTALITY AND MORBIDITY

84–1370. Fessel, M. **Mehrsterblichkeit von Rauchern. [Excess Mortality of Smoking.]** *Lebensversicherungs Medizin* 4(34): 79-81, May 4, 1982, German.

Results of eight prospective epidemiological studies showing excess mortality of smokers compared with nonsmokers are discussed along with mortality investigations of an American direct writing life insurance company and a European reassurance company. The extent of excess mortality varies considerably and depends on such parameters as age, amount smoked, duration of smoking, and form of tobacco smoked. Causes of death, smoking as a "socioeconomic index," and the changes and problems arising from adequate life insurance underwriting of smokers are examined. (Auth. Abs. Mod.)

84–1371. Kristein, M. M. **40 Years of U.S. Cigarette Smoking and Heart Disease and Cancer Mortality Rates.** *Journal of Chronic Diseases* 37(5): 317-323, 1984.

In a review of the association between cigarette smoking and mortality, results are reported on time series correlation studies of the relationship between per capita cigarette smoking and mortality rates for heart disease and respiratory cancer in the United States from 1940 to 1979. Major findings support the view that there is a 20-year lag relationship between smoking (especially consumption level) and respiratory cancer mortality, but no apparent lag in the relationship of smoking (especially pounds of tobacco consumed per capita) with heart disease mortality. Smoking explains over 90 percent of the variance in the relationship between smoking and respiratory cancer mortality, but explains only 50 percent of the variance between smoking and heart disease mortality. Findings dispute a 1981 report (*Journal of Chronic Diseases* 34(2/3): 87-103, 1981) that suggested warnings about the lethality of smoking issued by the Royal College of Physicians and the U.S. Surgeon General are precipitate and unwarranted. (Auth. Abs. Mod.) See also 81-0758.

84–1372. Millar, W. J. **Sex Differentials in Mortality by Income Level in Urban Canada.** *Canadian Journal of Public Health* 74: 329-334, September-October 1983.

Analysis of mortality data from census tracts in 21 metropolitan areas of Canada (ranked by median income of the tract) shows that mortality rates were higher for males compared with females in all income groups, but males in the lowest income level had the highest rates for all causes. While interpretation of sex differentials by specific cause of death is difficult because data on the distribution of known risk factors is lacking, in the case of ischemic heart disease, lung cancer, and emphysema, differences are consistent with the socioeconomic distribution of smoking patterns. Historically, regular smoking has been more prevalent among males and in the lowest income group. Females in the lowest income group also smoke more than those at higher levels. It is noted that low-income females are at greater risk of death for several causes than males in the highest income group.

84–1373. Smith, A. H.; Pearce, N. E. **Determinants of Differences in Mortality Between New Zealand Maoris and Non-Maoris Aged 15-64.** *New Zealand Medical Journal* 97(750): 101-108, February 22, 1984.

Maori mortality between 1974 and 1978 was much higher compared with other New Zealanders aged 15 to 64, and the higher death rates may be attributable to smoking. More than 50 percent of Maori males and 50 percent of Maori females over age 20 smoke cigarettes, a much higher rate than for non-Maori New Zealanders. Daily cigarette consumption levels are also higher among Maoris. The relative mortality risks for both sexes are much higher than expected for smoking-related respiratory diseases such as lung cancer, chronic bronchitis, and emphysema. The excess of smoking-related deaths among Maoris may be due to differences in smoking behavior, such as inhaling more or commencing to smoke at a younger age, or it may reflect differences in susceptibility to cigarette smoke. For coronary heart disease, the proportion of excess mortality among male Maori (beyond that linked to social class) that could be attributed to smoking was 15 percent; for females, it was 16 percent. It is concluded that higher mortality rates among Maoris result from a combination of factors, including cigarette smoking, socioeconomic status, alcohol consumption, and obesity.

See also, **84–1439, 84–1456, 84–1494**

NEOPLASTIC DISEASES

84–1374. Armstrong, R. W.; Armstrong, M. J.; Yu, M. C.; Henderson, B. E. **Salted Fish and Inhalants as Risk Factors for Nasopharyngeal Carcinoma in Malaysian Chinese.** *Cancer Research* 43: 2967-2970, June 1983.

While no association was found between the use of tobacco, alcohol, or nasal ointments and the incidence of nasopharyngeal cancer among Malaysian Chinese, any history of salted fish consumption during childhood was a significant risk factor (relative risk 3.0). A case—control study of 100 Malaysian Chinese (65 males, 35 females) and 100 matched controls was conducted to test inhalants, salted fish consumption, and use of tobacco and alcohol as risk factors. Childhood daily consumption of salted fish compared with nonconsumption carried a relative risk of 17.4. Occupational exposure to smoke and dust was also significantly associated with nasopharyngeal cancer. Salted fish consumption and occupational exposures were independent risk factors. (Auth. Abs. Mod.)

84–1375. Axelson, O. **Experiences and Concerns on Lung Cancer and Radon Daughter Exposure in Mines and Dwellings in Sweden.** *Zeitschrift fur Erkrankungen der Atmungsorgane* 161(6): 232-239, 1983.

Investigations of radon daughter exposure in homes revealed a synergistic relationship with smoking in lung cancer etiology among miners. Previous measurements had indicated that brick and concrete houses with basements (especially those located in areas with uranium-containing alum shale or granite) had significantly higher average radon levels than wooden houses without basements. During a 1967 to 1977 study period, lung cancer cases and referents were surveyed. Subjects were limited to those who had lived at the same address for 30 years prior to death. Smoking histories were available for half of the cases. In the high-exposure housing categories, smokers had a lung cancer crude risk ratio of 8.3, while in the low-exposure housing group the ratio was only 2.0, indicating a synergistic effect between smoking and radon daughter exposure. Residential exposure to radon daughters would explain the "urban" factor in lung cancer rates, and it is also recommended that low level radon daughter exposure be considered in future investigations of passive smoking. Previous studies of lung cancer among miners are reviewed.

84–1376. Axelson, O. **Room for a Role for Radon in Lung Cancer Causation?** *Medical Hypotheses* 13(1): 51-61, January 1984.

Smoking is a major cause of lung cancer, but the hypothesis of a synergistic effect of cigarette smoke and radon progeny would explain many of the inconsistencies observed in the simple model of a direct relationship between lung cancer and smoking. Radon daughters escape from stone building materials and the ground, and exposure has recently grown because of the trend toward central heating and increased indoor worktime. It is noted that ambient electrically charged radon progeny attach to available aerosol particles such as cigarette smoke. Such factors could explain geographical, urban-rural, and male-female differences in lung cancer rates. The possible synergism would also account for the lower lung cancer rates in tropical and subtropical areas and the familial factor in lung cancer etiology. There is also some evidence that radon daughter exposure is the main initiator, while smoking acts as a promoter of lung cancer. Radon exposure relationship with cancer among passive smokers and the role of smoking-related bronchitis in protecting against radon daughter-associated lung cancer are also discussed.

84–1377. Bartsch, H.; O'Neill, I. K. **Eighth International Meeting on *N*-Nitroso Compounds. Occurrence, Biological Effects, and Relevance to Human Cancer.** *Cancer Research* 44(3); 1301-1304, March 1984.

The formation and analysis of *N*-nitrosamines in tobacco products and their endogenous formation in tobacco consumers were reviewed in papers presented at a recent conference on *N*-nitroso compounds (NOC). It was reported that (1) NOC occur in fermented tobacco and tobacco smoke and represent the greatest and most widespread human exposure known at present; (2) concentration of NOC, especially tobacco-specific nitrosamines, is related to the nitrate content of tobacco products; (3) epidemiological evidence shows no important difference in risk due to the method of curing tobacco; (4) 4-(N-methyl-N-nitrosamino)-1-(3-pyridyl)-1-butanone (NNK) is a potent carcinogen that can cross the placental barrier in animals; (5) metabolic studies in vitro and in experimental animals indicate that NNK and *N'*-nitrosonornicotine readily convert into electrophiles that can interact with DNA; (6) nitrosation of betel nut constituents leads to the formation of 3 NOC, yielding carcinogens that could play a role in oral cancer development in betel quid chewers; and (7) an association has been confirmed between human cancer and tobacco chewing and snuff dipping practices in some southern States.

84–1378. Berggren, G.; Sjostedt, S. **Preinvasive Carcinoma of the Cervix Uteri and Smoking.** *Acta Obstetricia et Gynecologica Scandinavica* 62(6): 593-598, 1983.

The prevalence of smoking was 66.3 percent among 609 women with confirmed preinvasive cervical cancer compared with 41.9 percent among 6,090 controls ($p<0.001$). In addition, cancer patients had smoked longer, and average daily consumption was higher than controls. Relative risk was higher among women from rural areas, unmarried, and in the younger age groups. Subjects were women in Ostergotland County, Sweden, in whom a diagnosis of carcinoma in situ or severe dysplasia had been confirmed and reported to the Swedish Cancer Registry since 1977. Smokers were defined as those who had ever smoked regularly, including ex-smokers.

84–1379. Cann, C. I.; Fried, M. P. **Determinants and Prognosis of Laryngeal Cancer.** *Otolaryngologic Clinics of North America* 17(1): 139-150, February 1984.

Study data have shown that smoking and alcohol consumption are strongly associated with laryngeal cancer. In veterans consuming >40 cigarettes/day, the age-adjusted mortality rate was 15/100,000 person-years compared with a rate of only 0.6/100,000 for nonsmokers. Other studies have reported tobacco-related rates of 11 to 14/100,000. Tobacco and alcohol show a synergistic interaction in laryngeal cancer etiology, with a weighted average index of synergy of about 2.5. Smoking is also a confounding factor in occupational studies. Laryngeal cancer constitutes 2.3 percent of all cancers diagnosed in males and 0.4 percent in females. There has been a small age-specific increase in rates among white males aged 65 or older, a greater increase among black males (probably due to occupational factors), and an 80 percent jump in rates among women. This increase parallels the increase in lung cancer incidence rates, and is probably due to changes in smoking habits among women. The occurrence of additional primary cancers; relative survival rates; incidence and mortality data by age, sex, and ethnic group; and the risks associated with diet and occupation are also discussed.

84–1380. Chen, V. W.; Correa, P.; Zavala, D. E. **Lung Cancer: Leading Cause of Cancer Deaths in Louisiana White Females. (Letter).** *Journal of the National Cancer Institute* 72(1): 1-2, January 1984.

Recent data showing that lung cancer is now the leading cause of cancer deaths among white females living in Louisiana may be related to a high prevalence of smoking. When compared with the national average, lung cancer rates for white females in Louisiana showed a 10 percent excess between 1950 and 1969, and a 15.7 percent excess between 1968 and 1978. An even more pronounced excess (24.7 percent) was noted for white females between 1974 and 1977. A higher prevalence of smokers, more use of nonfilter cigarettes, and earlier smoking onset were observed in south Louisiana when compared with north Louisiana, where the prevalence of smoking appeared similar to that reported for a representative national sample.

84–1381. Craddock, V. M. **Nitrosamines and Human Cancer: Proof of an Association?** *Nature* 306(5944): 638, December 15, 1983.

Reports from a recent international meeting on *N*-nitroso compounds suggest that proof of an association between tobacco smoke nitrosamines and human cancer has been found. Epidemiological research shows an association between oral cancer in the U.S. southern States and snuff-dipping (as distinct from snuff sniffing or tobacco chewing), which involves ingestion of large amounts of nitrosamines. Nitrosamine levels in the environment have also been found sufficient to cause cancer. Analyses in regions with dramatic differences in incidence of esophageal cancer and in areas with a high incidence of stomach cancer, however, have not revealed any simple correlation between exposure and cancer incidence. This finding may be due to the many factors involved in disease etiology, leading researchers to examine variations in nitrosation between individuals, methods of determining such variations via urine analysis, and the effect of diet on susceptibility of host tissues. To unravel cancer's multifactorial etiology, it will be necessary to determine the molecular mechanisms by which nitroso compounds act. Preliminary findings from an animal experiment, which involved treating rodents with 15 dose levels of various nitrosamines for life, showed clearly measurable effects at doses of 0.01 mg/kg/day, emphasizing the need for preventive measures to lower nitrosamine levels in tobacco products, food, and the environment.

84–1382. Garcia, E. Y. **Can This Be the Way Tobacco Smoke Cause Lung Cancer?** Laguna, the Philippines, The Medical Research Clinic, 1982, 13 pp.

In an attempt to establish the link between tobacco smoke and lung cancer, a pipe-smoking doctor studied the progression of the disease in himself and conducted experiments in rats, with tar collected from the pipe filter. Results indicate that (1) nicotine is the carcinogen in tobacco smoke tar and is a complete nonprotein nitrogen with tissue affinity for mammals; (2) upon entry (facilitated by adrenalin) to the bronchial epithelial (target) cells, nicotine will be met by hostile RNAs and DNAs that will synthesize massive numbers of anticarcinogens, which in turn will synthesize ribonuclease and desoxyribonuclease that can deactivate the adverse effects of nicotine on the bronchial epithelial cells; (3) if the nicotine molecules increase geometrically, the target cells will also multiply geometrically to cope; and (4) if multiplication of the target cells goes unabated, they will become dynamic metaplastic and anaplastic cells (cancer cells) till death of the patient. (Auth. Abs. Mod.)

84–1383. Gazit, D.; Ulmansky, M.; Fischman, S.; Bab, I.; Sela, J. **A Study of a Sample of Oral Cancer in Israel.** *Oral Surgery, Oral Medicine, Oral Pathology* 57(1): 118-121, January 1984.

Although the prevalence of cigarette smoking is high among Israelis, alcohol abuse is low, which may account for the relatively stable incidence of oral cancer in Israel (currently somewhat lower than in other countries). Oral cancer represents 2.5 percent of all cancers, and is usually diagnosed in the seventh or eighth decade of life (43.5 percent of cases are seen in patients over age 71). Data compiled in 1977 show that 45 percent of those under age 29, 43 percent of those aged 30 to 39, 32 percent of 40- to 54-year-olds, and 28 percent of those over age 55 are regular cigarette smokers. Of these, 26 percent consume 15 to 20 cigarettes/day and 35 percent consume over 21 cigarettes/day. Thus, the low incidence of alcohol abuse, postulated to be synergistic with tobacco use in oral cancer etiology, may explain the fewer cases of oral neoplasms.

84–1384. Glantz, S. A. **What To Do Because Evidence Links Involuntary (Passive) Smoking With Lung Cancer.** *Western Journal of Medicine* 140(4): 636-637, April 1984.

Data from recent research on the health effects of passive smoking indicate a link between involuntary smoking and lung cancer. Such findings have generated serious concern in the tobacco industry because they threaten to change the established social environment that has been conducive to promoting tobacco products. Nonsmokers are also motivated to take a more active stand on the smoking issue. The industry has systematically attempted to undermine the scientific link between smoking and disease, and is now spending millions of dollars to point out inconsistencies in findings of studies on passive smoking and lung cancer published in 1981. However, recent research that examined all cancer deaths among nonsmoking wives in northwestern Pennsylvania indicates a general and significant association between passive smoking and risk of death due to cancer. Individual physicians are urged to take simple, inexpensive steps to protect nonsmokers from the adverse health effects of passive smoking in medical facilities and to support legislative efforts protecting nonsmokers in the workplace and other public settings.

84–1385. Greenberg, E. R.; Korson, R.; Baker, J.; Barrett, J.; Baron, J. A.; Yates, J. **Incidence of Lung Cancer by Cell Type: A Population-Based Study in New Hampshire and Vermont.** *Journal of the National Cancer Institute* 72(3): 599-603, March 1984.

Review of medical records for 1,486 lung cancer patients in Vermont and New Hampshire indicates that the overwhelming majority of cases had been smokers, regardless of sex or cancer cell type. Relatively fewer women and adenocarcinoma cases were smokers. Among women, the incidence rate for small cell undifferentiated cancer was about equal to that for squamous cell carcinoma, while among men, small cell undifferentiated carcinoma occurred much less often than squamous cell carcinoma. Findings support the suggestion that smoking disproportionately increases the risk of small cell undifferentiated carcinoma relative to other cell types in women, but not in men.

84–1386. Harley, N. H. **Radon and Lung Cancer in Mines and Homes.** *New England Journal of Medicine* 310(23): 1525-1527, June 7, 1984.

Inconsistencies in epidemiological findings concerning the etiology of lung cancer with regard to smoking and radon daughter exposure are analyzed. Exposure data, measured by alpha dosimetry and expressed in working-level months (WLM), indicate that among Swedish miners with equivalent exposure to radiation, excess cases of lung cancer were greater in nonsmokers than in smokers. A study of Navajo Indian (among whom smoking is uncommon) lung cancer patients showed that uranium miners were an average of 20 years younger than nonminers, and confirmed the excess risk of that group. It is estimated that the average environmental exposure of miners is 0.2 WLM/year, lifetime exposure is 15 WLM, and the projected lung cancer risk is 15 cases/1,000 persons. This finding is inconsistent with the observed nonsmokers' risk of <10 cases/1,000. Current research indicates that radon daughter-induced lung cancer accounts for about 20 percent of the "spontaneous" lung cancer in nonsmokers.

84–1387. Hoch-Ligeti, C.; Sass, B.; Sobel, H. J.; Stewart, H. L. **Endocardial Tumors in Rats Exposed to Durable Fibrous Materials.** *Journal of the National Cancer Institute* 71(5): 1067-1071, November 1983.

Endocardial tumors in the rat found in association with intrathoracic implantation of durable fibrous materials, including cigarette smoke condensate plus crocidolite fibers, are reported for the first time. In Osborne-Mendel female rats, endocardial tumors occurred in 1 of 15 rats. The endocardial tumor lining the cardiac chambers was contiguous with tumor invading the wall of the right ventricle and was visible to the unaided eye. The invading tumor cells were elongated or polygonal and infiltrated interstitially and into the heart muscle fibers. Intrathoracic administration of pulverized chrysotile, coarse cotton lint, and intravenous injections of air particulate extract were each associated with 1 endocardial tumor in 15 rats. Tumors occurred in 3 of 50 rats with fiberglass implants

>5 µm in length and 1 to 3 µm in diameter, in 1 of 15 rats with fiberglass implants of 0.03x3 µm, and 1 of 15 rats with implants of pulverized glass fiber. In all cases, the dimensions of durable fibers associated with endocardial tumors were similar to those of carcinogenic asbestos. Endocardial tumors were not observed in concurrently studied control rats. (Auth. Abs. Mod.)

84–1388. Kantor, A. F.; Hartge, P.; Hoover, R. N.; Narayana, A. S.; Sullivan, J. W.; Fraumeni, J. F., Jr. **Urinary Tract Infection and Risk of Bladder Cancer.** *American Journal of Epidemiology* 119(4): 510-515, April 1984.

Evidence of an interactive effect between urinary tract infection and cigarette use was observed in an epidemiologic study of 2,982 bladder cancer patients and 5,782 population controls from 10 geographic areas of the United States. The role of urinary tract infection and inflammation in the etiology of this neoplasm was specifically evaluated. A history of urinary tract infection significantly elevated the risk of bladder cancer, particularly in individuals (nonsmokers) who reported three or more infections (relative risk (RR) = 2.0). The joint effect of smoking and infection was slightly beyond that expected under an additive (but not multiplicative) model, with the exception of subjects in the extreme exposure categories. Among smokers of < 20 cigarettes/day, who had three or more infections, the RR was 4.3; for smokers of 20 to 39 cigarettes/day, 5.6; and for ≥ 40 cigarettes/day, 2.8. Significantly increased bladder cancer risk was also found for bladder stones (RR = 1.8), while the occurrence of kidney stones showed no relationship. A history of three or more urinary tract infections was strongly related to squamous cell carcinoma in particular (RR = 4.8). The moderate interaction between urinary infection and cigarette use suggests that patients with cystitis may be especially prone to tobacco-derived carcinogens in the urine. (Auth. Abs. Mod.)

84–1389. Kinlen, L. J.; McPherson, K. **Pancreas Cancer and Coffee and Tea Consumption: A Case—Control Study.** *British Journal of Cancer* 49(1): 93-96, January 1984.

Examination of data from a case—control study of 216 pancreatic cancer patients (109 males, 107 females) conducted in northwest England and north Wales in the early 1950s showed a positive relationship between smoking and pancreatic cancer. There was a higher, but not statistically significant risk associated with smoking ≥ 50 cigarettes/week (relative risk (RR) = 1.66) than with smoking 10 to 49 cigarettes/week (RR = 1.26). RRs were similar for both sexes. No significant relationship between smoking habits and coffee consumption was found, although among women, more heavy tea drinkers (23 percent) smoked than did moderate tea drinkers (17 percent). No positive relationship between coffee drinking and pancreatic cancer was found.

84–1390. La Vecchia, C.; Franceschi, S. **Italian Lung Cancer Death Rates in Young Males. (Letter).** *Lancet* 1(8373): 406, February 18, 1984.

The urgent need for modification of smoking habits in Italy is stressed, since lung cancer mortality (the leading cause of cancer mortality) in young men (aged 35-44) is significantly higher compared with rates in many developed countries (except the United States). Current cancer rates are of crucial importance in determining trends among older men in future decades. In 1980, 54.3 percent of males in Italy were current smokers; 64.9 percent at ages 30 to 39, 61.7 percent of those aged 40 to 59, and 52 percent of those aged 60 to 70. In those aged 60 to 70, 23.1 percent were ex-smokers, and in those over 70 years of age, 38.6 percent were smokers and 28.1 percent were ex-smokers. If a massive epidemic of lung cancer at the beginning of the 21st century is to be avoided, when British and American rates are expected to be falling, practical actions are required, such as increasing the price of cigarettes.

84–1391. Meyer, M. B.; Luk, G. D.; Sotelo, J. M.; Cohen, B. H.; Menkes, H. A. **Hypothesis: The Role of the Lung in Stomach Carcinogenesis.** *American Review of Respiratory Disease* 121(5): 887-892, May 1980.

It is proposed that pulmonary defense mechanisms against cigarette smoke play an important role in stomach carcinogenesis. Inhaled carcinogens reach the stomach by normal pulmonary clearance and account for the association of stomach cancer with dusty occupations. It is postulated that although familial and other factors determine the susceptibility to endothelial cancer (both stomach and lung), persons whose pulmonary clearance mechanisms are more susceptible to impairment by cigarette smoke retain carcinogens and other particulate matter in the lungs and are at increased risk for lung cancer. Those whose clearance mechanisms are not impaired continue to clear these particles from their lungs, unconsciously swallow the cleared particles, receive more of these substances in the stomach, and are at increased risk for stomach cancer. This would account for (1) the striking dose—response relationship between the amount of smoking and lung cancer mortality and the relatively weak relationship between smoking and stomach cancer; and (2) the observed reciprocal relationships in time trends of mortality between stomach cancer and lung cancer. (Auth. Abs.)

84–1392. Miller, G. H. **Cancer, Passive Smoking and Nonemployed and Employed Wives.** *Western Journal of Medicine* 140(4): 632-635, April 1984.

Mortality due to cancer showed an almost twofold increase among nonemployed wives with long-term exposure to passive smoking compared with nonemployed wives who experienced little or no exposure. Data on cause of death, smoking history, and other relevant information were obtained in Erie County, Pennsylvania, in 1975-1976. Nonsmoking wives (n = 547) were classified by husband's smoking history and their own employment outside the home. Data showed a highly positive, but not statistically significant, association between long-term passive smoking and cancer as a cause of death for the entire group. The odds ratio was 0.40 and the χ^2 value was 2.18 for all wives. When women working outside the home were omitted, the odds ratio reached significance at 1.94. When working wives were included, the odds ratio for women aged 59 and younger was high and statistically significant. Findings support the hypothesis that long-term passive smoking leads to excess cancer deaths in exposed nonsmokers. For employed wives, the fact that husbands smoked or did not smoke seemed unimportant, probably because of exposure to smoke and other hazards in the workplace.

84–1393. Nahas, F.; Lichtig, C.; Gershon, H. **In Vitro Induction of Malignant Transformation and In Vivo Resistance to Tumor Growth in the X/Gf Mouse.** *Journal of the National Cancer Institute* 71(1): 217-221, July 1983.

Using the chemical carcinogens 7,12-dimethylbenz(α)anthracene and benzo(*a*)pyrene as inducers, sarcomas were produced in cloned transformed cells of tumor-resistant X/Gf mice. Mice produced by breeding the X/Gf strain with a tumor-susceptible strain (C57BL/6J) were more susceptible than the resistant parent, but less susceptible than the susceptible parent. It was thus established that the tumor resistance of the X/Gf mouse is not based on resistance of isolated cells of these animals to malignant transformation, but rather is attributable to an in vivo homeostatic mechanism that prevents tumor growth.

84–1394. Orbuch, S. J.; Perazzo, D. L.; Porta, J.; Garrido, C. **Carcinoma Epidermoide de Pulmon en Gemelos Univitelinos. [Epidermoid Carcinoma of the Lung in Identical Twins.]** *Medicina* 43(1): 16-20, 1983, Spanish.

Epidermoid cancer of the lung developed in a pair of identical twins, both heavy smokers (40 and 60 cigarettes/day, respectively). Cytogenetic studies revealed a translocation in chromosome group D in the twins, their mother, and four of five children of one twin. HLA haplotyping was done on the twins and their families in an attempt to examine the interaction between environmental carcinogens and susceptible genotypes. It is concluded that the simultaneous appearance of lung cancer in the twins in the presence of heavy tobacco use is an example of ecogenetics.

84–1395. Pastorino, U.; Berrino, F.; Gervasio, A.; Pesenti, V.; Riboli, E.; Crosignani, P. **Proportion of Lung Cancers Due to Occupational Exposure.** *International Journal of Cancer* 33: 231-237, February 1984.

Smoking contributed 81 percent of attributable risk of lung cancer in a population-based case—control study in an industrialized area of northern Italy from 1976 to 1979. Two hundred and four of the 211 lung cancer cases and 351 controls from the general population were questioned about occupational and smoking histories. Subjects were classified as probably exposed, possibly exposed, or unexposed to the following chemicals, known to be carcinogenic: asbestos, polycyclic aromatic hydrocarbons, arsenic, nickel and chromium compounds, bis-chloromethyl-ether, chloromethyl-methyl ether, and vinyl chloride. Lung cancer attributable to occupational exposure was 33 percent; to occupational exposure and smoking combined, 89 percent. In subjects exposed to occupational carcinogens, the risk fraction preventable by reducing tobacco only is 38.8 percent; the fraction preventable by acting on occupational risk only, 18.8 percent. (Auth. Abs. Mod.)

84–1396. Radford, E. P.; Renard, K.G.S.C. **Lung Cancer in Swedish Iron Miners Exposed to Low Doses of Radon Daughters.** *New England Journal of Medicine* 310(23): 1485-1494, June 7, 1984.

The effects of smoking and exposure to alpha radiation from radon daughters were nearly additive in a retrospective investigation of lung cancer mortality from 1951 to 1976 in 1,415 Swedish iron miners who had received annual doses close to the currently accepted occupational limits. Fifty deaths from lung cancer were observed compared with 12.8 expected; expected rates were determined by a smoking-specific analysis based on data from a random sample of the Swedish male population. Among nonsmokers, 18 deaths occurred compared with 1.8 expected; among current smokers and recent ex-smokers, 32 deaths were observed and 11.0 expected. Comparison of lung cancer risk coefficients from this and other cohort studies of underground miners showed good agreement. Exposure to radon daughters is a major medical problem in underground metal mining, but results also indicate that exposure to radon daughters at home accounts for an appreciable number of cases of lung cancer in the general population. (Auth. Abs. Mod.)

84–1397. Reizenstein, P. **Female Superiority.** *Biomedicine and Pharmacotherapy* 36(6/7): 275-276, 1982.

There are indications that women are more resistant than men to malignant transformation induced by radiation or smoking. Studies of dose—response malignant mortality among women do not show an advantage over men. However, the number of bronchial metaplasias is significantly higher in male than in female smokers who consume an equal number of cigarettes. Three possibilities are suggested to explain the difference: (1) exposure to carcinogens other than those under study is lower in women; (2) the difference is due to the presence of hormone receptors on tumor cells, as has been observed in leukemia and where female sex is an indicator for good prognosis; and (3) the difference is related to survival expectancy, which is 79 years and still increasing in women, while male life expectancy has remained static at 73 years for the past decade.

84–1398. Riboli, E.; Bai, E.; Berrino, F.; Merisi, A. **Mortality From Lung Cancer in an Acetylene and Phthalic Anhydride Plant. A Case—Referent Study.** *Scandinavian Journal of Work, Environment and Health* 9(6): 455-462, December 1983.

A case—referent study of occupational exposure to acetylene and phthalic anhydride at an Italian chemical plant (SISAS) revealed a crude odds ratio (OR) of 76.0 for lung cancer among occupationally exposed heavy smokers. Few subjects were nonsmokers, thus the referent group combined nonsmokers with light (<10 cigarettes/day) smokers. Cases were 43 males who died of lung cancer from 1976 to 1979; age-matched referents ($n=99$) died of other causes during the same period. Smoking, occupational history, and validation of cause of death were obtained by interview with next of kin. For light smokers, the crude OR was 14.3 for SISAS-exposed cases, 2.1 for other occupational exposures, and 1.0 for nonexposure. For moderate smokers (10-19 cigarettes/day), ORs were 12.7, 8.9, and 4.8, respectively, and for heavy smokers (≥ 20 cigarettes/day), 76.0, 21.1, and 13.8, respectively. After controlling for age and smoking, the OR for SISAS-exposed subjects relative to nonexposed subjects was 5.6. The risk for exposure in other work environments was 1.7. Because of the few cases in each category, confidence intervals were very broad. About one-third of lung cancer risk was attributable to occupational exposure to chemical carcinogens.

84–1399. Rinaldi, M.; Tonachella, R.; Nardi, M.; Venturo, I.; Gallo Curcio, C. **Valutazione dei Risultati di Un'Indagine su 343 Carcinomi Polmonari Desunti da una Scheda Rilevamento Dati. [Evaluation of Epidemiological Data From 343 Lung Cancer Patients Drawn From a Protocol Sheet.]** *Recenti Progressi in Medicina* 74(6): 678-685, June 1983, Italian.

Epidemiological data, which included smoking habits, age, occupation, demographics, symptoms, and site and histological type of primary lesion, were evaluated for 343 cases (324 men and 19 women, 95.3 percent smokers) of inoperable lung cancer. Of the 16 nonsmokers, 12 were women. Among the smokers, 2.7 percent smoked <10 cigarettes/day, 29.9 percent smoked 10 to 20 cigarettes/day, and 67.2 percent >20/day. Only 125 patients reported smoking nonfiltered cigarettes; 243 smoked filtered products. Among the 220 heavy smokers, 90 had squamous cell, 79 undifferentiated, 29 small cell, and 22 adenocarcinomas. The most frequently reported "alarming symptoms" were chest pain (27.7 percent) and cough (20.9 percent).

84–1400. Samet, J. M.; Kutvirt, D. M.; Waxweiler, R. J.; Key, C. R. **Uranium Mining and Lung Cancer in Navajo Men.** *New England Journal of Medicine* 310(23): 1481-1484, June 7, 1984.

Results demonstrate that in a rural nonsmoking population, most of the cases of lung cancer may be attributable to one hazardous occupation. Of the 32 Navajo men with lung cancer diagnosed between 1969 and 1981, 23 had been employed as uranium miners. Smoking habit information for 21 of the 23 showed that 8 were nonsmokers and the rest smoked less than 8 cigarettes/day. The population-based case—control study examined the association between uranium mining and lung cancer. For each case in a Navajo man, two controls with nonrespiratory cancer and no documented experience in the uranium industry were selected from the New Mexico Tumor Registry. Because smoking histories were unavailable for controls, the relative risk associated with cigarette smoking could not be directly assessed. Further followup will be needed for a complete characterization of the consequences of uranium mining in Navajos.

84–1401. Stevens, R. G.; Moolgavkar, S. H. **A Cohort Analysis of Lung Cancer and Smoking in British Males.** *American Journal of Epidemiology* 119(4): 624-641, April 1984.

Analysis of lung cancer mortality in England and Wales among males aged 25 to 84 showed that, from 1941 to 1975, 88 percent of total deaths could be attributed to smoking. Using cigarette consumption information from the Tobacco Research Council and a statistical model, relative and attributable risks were estimated. Whereas the mortality rate among smokers increased over the entire span of the analysis, the rate among nonsmokers rose gradually until 1956 to 1960 and then declined until 1971 to 1975. The beginning of the decline coincided with the implementation of the Clean Air Act of 1956. Monte Carlo simulations were performed to address possible systematic biases in the smoking data. (Auth. Abs. Mod.)

84–1402. Temeck, B. K.; Flehinger, B. J.; Martini, N. **A Retrospective Analysis of 10-Year Survivors From Carcinoma of the Lung.** *Cancer* 53(6): 1405-1408, March 15, 1984.

Of 4,732 lung cancer patients seen between 1949 and 1972, 118 (91 men, 27 women; 86 percent smokers) were found alive 10 years after initial diagnosis and treatment. The median age was 57 years. Among the 115 who had surgery, 18 also had postoperative external radiation. The case—control study was designed to evaluate the importance of stage, symptoms, smoking history, and family history as predictors of 10-year survival in patients matched by histology, age, sex, and year of diagnosis with those who lived less than 2 years after treatment. Although the vast majority of both groups of patients were smokers (101/118 10-year survival, 100/118 ≤ 2-year survival), neither smoking history nor family history appeared to have any discriminating power, and symptoms seemed to be important only as a reflection of stage. Results indicated that stage at diagnosis was an overwhelmingly important factor. No long-term survivor died of the original lung cancer, suggesting that 10-year survival is adequate proof of cure of primary cancer of the lung.

84–1403. Teta, M. J.; Walrath, J.; Meigs, J. W.; Flannery, J. T. **Cancer Incidence Among Cosmetologists.** *Journal of the National Cancer Institute* 72(5): 1051-1057, May 1984.

In a retrospective study of 13,650 Connecticut cosmetologists licensed ≥ 5 years, a strong association between the year they first attended hairdressing school and cancer incidence was found, consistent with the observation that smoking prevalence in the 1930s was much higher among hairdressers than in the general population. Total cancer incidence was determined from 1938 to 1982, and observed to expected cases (O:E ratio) were excessive for respiratory, ovarian, and brain cancer in women. Rates in men were close to expected. In females, the O:E ratio of smoking-related cancers was 111:78.88 ($p < 0.001$); in the 1925-1934 entry cohort, the O:E ratio was 57:32.08 ($p < 0.0001$). This excess of smoking-related cancers accounted for more than 40 percent of the excess incidence among women. Reproductive histories were not available, preventing analysis of the excess of ovarian cancer. No specific occupational agent could be identified.

84–1404. Wiman, L.-G.; Nilsson, B.; Bylin, G. **Lungcancer i Stockholms-Regionen — Arlig Incidens 1958—80, Prognos 1981—85. [Lung Cancer in the Stockholm Region — Incidence and Prognosis.]** *Lakartidningen* 36: 3290-3293, 1983, Swedish.

Changes in smoking habits are suggested as the most likely explanation for the new pattern of lung cancer incidence in Stockholm. Age-adjusted incidence rates for lung cancer were studied for both men and women and separately for histological subtypes of cancer, and a 5-year prognostic model was produced. Results show a decrease in incidence for men from 1973 and a steady increase for women over the entire study period from 1958 to 1980. The prognostic trend for 1981 through 1985 indicates a continuing decrease in incidence for men and an increasing incidence among women.

NON—NEOPLASTIC RESPIRATORY DISEASES

84–1405. Agnew, J. E.; Bateman, J.R.M.; Pavia, D.; Clarke, S. W. **Radionuclide Demonstration of Ventilatory Abnormalities in Mild Asthma.** *Clinical Science* 66(5): 525-531, May 1984.

No relationship between lung function and smoking habit was found among asthmatics when the distribution of 5 µm ^{99m}Tc aerosol particles deposited in the lungs was compared with that of ^{81m}Kr gas in 20 mild or asymptomatic asthmatics (5 smokers, 5 ex-smokers, 10 nonsmokers). Controls were 16 normal nonsmokers and 14 "normal" cigarette smokers. Radioaerosol lung penetration in normal smokers was the same as in normal nonsmokers, but was reduced in asthmatic subjects. Radioaerosol penetrated better than ^{81m}Kr to the lung apices of normal nonsmokers and asthmatic subjects and to the lung bases of asthmatic subjects. Uneven distribution of aerosol relative to ^{81m}Kr related strongly to maximal mid-expiratory flow rate in asthmatic subjects. Aerosol deposition abnormalities in symptom-free asthmatic subjects may reflect basal airways closure at functional residual capacity and abnormal patterns of lung filling. (Auth. Abs. Mod.)

84–1406. Bardana, E. J., Jr.; Main, D. M.; Hogan, T. J. **Effect of Cigarette Smoke on Formaldehyde Data. (Letters).** *Journal of Occupational Medicine* 26(6): 410-412, June 1984.

A previous study (*Journal of Occupational Medicine* 25(12): 896-900, December 1983) that linked respiratory symptoms to formaldehyde exposure is criticized because (1) nearly 80 percent of both "exposed" and "nonexposed" workers (policemen) were smokers; (2) tobacco smoke as a source of formaldehyde was not accounted for; (3) formaldehyde was not measured in the work areas of "nonexposed" policemen; and (4) allowance was not made for alternative chemicals, especially those in tobacco smoke. In reply, the authors state that exposure to mainstream smoke was about the same in both groups, while sidestream smoke exposure was probably higher in the control group (policemen confined to patrol cars) compared with the experimental group (policemen in the formaldehyde-contaminated office trailers). Also, formaldehyde readings in the trailers were taken after they had been evacuated for several months, and thus the observed levels of 0.12 to 1.6 ppm were not influenced by cigarette smoke. Other chemical agents, such as ozone, were considered and discarded because there were no sources. The formaldehyde-related increase in respiratory symptoms among policemen working in the trailers was still evident when groups were analyzed by smoking status, but because of the small numbers, results were not reported. See also 84-0786.

84–1407. Beaty, T. H.; Menkes, H. A.; Cohen, B. H.; Newill, C. A. **Risk Factors Associated With Longitudinal Change in Pulmonary Function.** *American Review of Respiratory Disease* 129(5): 660-667, May 1984.

Only age and smoking were consistently associated with increased loss of forced expiratory function in both men and women, tested twice, with an average of 4.7 years between visits. The relationship between loss of forced expiration over time and a series of potential risk factors and four measures of pulmonary function recorded at initial visit was investigated in 1,912 adults (1,019 men, 893 women). Statistically significant differences among ABO blood types and among Pi types were seen in women, although the direction of these differences was unexpected in that non-Z Pi variant phenotypes showed less decline in pulmonary function than did the most common Pi M phenotypes. Similar patterns of differences were seen in men, although these were not statistically significant. Of four initial-visit pulmonary function measures examined, only closing capacity at initial visit was consistently associated with subsequent loss of forced expiratory volume in 1 sec (FEV$_1$) in both men and women. However, the final predictive model for FEV_1 accounted for only a modest proportion of the total variation seen, limiting its potential use for predicting decline in pulmonary function over a 5-year span in individual subjects. (Auth. Abs. Mod.)

84–1408. Beck, G. J.; Schachter, E. N.; Maunder, L. R.; Schilling, R.S.F. **Cotton Dust and the Lung. (Letter).** *Annals of Internal Medicine* 100(1): 162, January 1984.

In responding to a previous letter (*Annals of Internal Medicine* 99(2): 278, August 1983), the authors of the original report on chronic lung disease in cotton textile workers state that the wrong tables were used to support the hypothesis that "nonsmoking cotton worker's impairment might regress after retirement." The data cited were for both active and retired workers, rather than only retired workers, and thus results claimed are irrelevant. The appropriate data support the conclusion that chronic lung disease in these workers may be irreversible. See also 84-0779, 84-0791, 84-0792.

84–1409. Donham, K. J.; Zavala, D. C.; Merchant, J. A. **Respiratory Symptoms and Lung Function Among Workers in Swine Confinement Buildings: A Cross-Sectional Epidemiological Study.** *Archives of Environmental Health* 39(2): 96-101, March-April 1984.

Smoking appeared to have an additive effect for symptoms of chronic bronchitis and chronic wheezing in subjects (n=24, 9 smokers) working in swine-feeding structures compared with swine workers (n=24, 9 smokers) not so confined. The cohort of confinement workers was matched for age, sex, and smoking history with nonconfinement swine producers. Pulmonary function studies and a survey questionnaire for chronic respiratory disease symptoms were performed on both groups. Compared with controls, confinement workers experienced significantly higher prevalence of chronic bronchitis and wheezing (odds ratio 7 and 4, respectively). There were, however, no significant differences in baseline pulmonary functions. Examination of combined chronic respiratory symptoms reinforced the relationship of smoking and confinement work. Nonconfinement nonsmokers had the lowest score (0.70); confinement workers who smoked had the highest (3.80). Based on the high prevalence of chronic respiratory disease symptoms, this study emphasizes an emerging occupational concern in agriculture for people working in swine confinement operations and those who work in poultry, veal, beef, or dairy confinement operations. It is concluded that a representative population of these workers should be studied prospectively to determine if a progressive loss in lung function is evident. (Auth. Abs. Mod.)

84–1410. Fairshter, R. D. **Pharmacologic Management of Chronic Obstructive Pulmonary Disease.** *Comprehensive Therapy* 10(3): 12-19, March 1984.

Cessation of smoking is fundamental to the treatment of chronic obstructive pulmonary disease (COPD), but smoking cessation clinics have success rates of only 15 to 25 percent at 1-year followup. Lobeline sulfate is ineffective, but nicotine chewing gum has been associated with a cessation rate of 47 percent, significantly higher than rates in a comparison placebo group (21 percent). Side effects of the gum were mild (hiccups, gastrointestinal complaints, tiredness from chewing). Other factors in COPD management, including bronchodilators, antimicrobial agents, pulmonary hypertension, and right ventricular dysfunction, are discussed.

84–1411. Flenley, D. C. **New Notions in Chronic Bronchitis and Emphysema.** *Practitioner* 227: 1681-1691, November 1983.

The recently introduced proteolytic theory of the pathogenesis of emphysema proposes that (1) alveolar macrophages cluster in the small terminal bronchioles deep within the lung lobules of smokers; and (2) cigarette smoke damages the macrophages, leading to changes in appearance on electron microscopy and stimulating release of a neutrophil chemotactic factor. Although animal experiments support the theory, numerous problems remain, and it is not yet clear why some people develop cigarette-induced emphysema at a relatively early age, while lifetime smokers may develop very little emphysema. Current research suggests that the pattern of inheritance of α_1-antitrypsin does not explain this phenomenon. Therapeutic implications include the search for drugs to inhibit elastase and for agents that may increase production of α_1-antitrypsin, but no substances safe for human use have yet been formulated. Cessation remains the treatment of choice, and the general practitioner can be an effective change agent. In addition to conducting clinical tests for objective evidence of the effects of smoking, the use of nicotine chewing gum could be recommended to help reduce withdrawal symptoms. To date, however, increased cigarette taxation appears the most effective approach for reducing smoking levels among the British.

84–1412. Freedman, S.; Raffin, T. A.; Rothkopf, M. H.; Eddy, D. M.; Macklem, P. T. **The Value of a Stage Prop: Screening for Chronic Obstructive Pulmonary Disease.** *Chest* 85(3): 406-408, March 1984.

The controversy over the proposed screening of all adult patients for chronic obstructive pulmonary disease (COPD) as part of routine physical examinations is discussed. Critics of such a practice argue that it would cost approximately 500 million dollars/year with few benefits. Current evidence indicates that smoking causes COPD and that smoking cessation is the only cure for small airways disease and accelerated forced expiratory volume losses. Physician advice would not be different even with a positive test result, since even a perfect test would add almost no new information about the risk of death in smokers. Only about 5 percent of deaths annually are due to causes (including COPD) other than cardiovascular disease and cancer. Physicians would have only an added reason for urging patients not to smoke. In response, an advocate of screening maintains that the practice would not be nearly as expensive as suggested, and recommends incorporating measurement of forced expiratory volume in 1 sec and forced vital capacity into routine clinical examinations at no cost to the patient. Thus, spirometry would become part of the physical examination in the same way as measurement of temperature and blood pressure. However, routine screening of populations in an epidemiologic survey to identify smokers at risk is not recommended.

84–1413. Goldstein, E. **Hydrolytic Enzymes of Alveolar Macrophages.** *Reviews of Infectious Diseases* 5(6): 1078-1092, November-December 1983.

The effects of smoking are included in a review of the hydrolytic enzymes of pulmonary alveolar macrophages. The general properties, physiologic function, cellular physiology, and clinical associations are summarized for lysozyme, elastase, plasminogen activator, and collagenase. It is noted that cigarette smoke stimulates elastase secretion, while the peroxidase in the smoke inactivates α_1-antiprotease. This upsets the delicate balance between the elastases and protease inhibitors, which is implicated in emphysema etiology. The role in the inflammatory process and function in the pulmonary defense system of each enzyme is also described.

84–1414. Hillerdahl, G.; Rylander, R. **Asthma and Cessation of Smoking.** *Clinical Allergy* 14(1): 45-47, January 1984.

Of 125 consecutive asthma patients surveyed, 59 had quit smoking permanently or for short periods of time, 11 were current smokers, and 55 were nonsmokers or had quit at least 1 year before onset of asthma symptoms. Of those who quit, 18 (30 percent) reported a worsening of symptoms after cessation, 3 of whom had no symptoms prior to cessation. In 21 patients, asthma symptoms declined, and in the remaining 20, no change in symptoms was reported following cessation. It is postulated that quitting smoking could bring about a worsening of asthma symptoms through an increased capacity to form antibodies, less efficient mucociliary clearance, or transitional disturbances in the immune system.

84–1415. Kagawa, J. **Exposure-Effect Relationship of Selected Pulmonary Function Measurements in Subjects Exposed to Ozone.** *International Archives of Occupational and Environmental Health* 53(4): 345-358, March 1984.

Nonsmokers ($n = 10$) were more affected than smokers ($n = 7$) when healthy male volunteers were exposed to various ozone (O_3) concentrations with and without intermittent light exercise. Mean specific airway conductance was decreased 9.69 percent of baseline among nonsmokers and 6.37 percent among smokers after exposure to 0.15 ppm O_3. The level of exercise was related to the magnitude of changes in lung function and occurrence of respiratory symptoms at any O_3 exposure level. It is concluded that the two important factors in evaluating the exposure—effect relationship of O_3 are level of exercise and smoking habit. (Auth. Abs. Mod.)

84–1416. Kawakami, Y.; Yamamoto, H.; Yoshikawa, T.; Shida, A. **Chemical and Behavioral Control of Breathing in Adult Twins.** *American Review of Respiratory Disease* 129(5): 703-707, May 1984.

In the 28 pairs of monozygotic (MZ) twins studied, it is assumed that genetic factors had influenced smoking habits and that the closer similarity of hypoxic ventilatory response was not a direct result of similar smoking habits, since smoking habits were more similar in MZ twins compared with the 10 pairs of dizygotic (DZ) twins. Mean values for hypoxic and hypercapnic ventilatory responses, threshold for perceiving added inspiratory resistance, and respiratory patterns were not different between MZ and DZ twins. However, within-pair variance ratios (those in MZ twins being denominators) for hypoxic response (4.08, $p < 0.005$), hypercapnic response (4.89, $p < 0.005$), respiratory frequency during air breathing (3.96, $p < 0.005$), inspiratory time during air breathing (5.47, $p < 0.005$), and inspiratory time during hypoxia (9.08, $p < 0.005$) were significantly larger than 1. Within-pair variances for threshold for resistive load perception and respiratory pattern during hypercapnia were equivalent between the two groups. Results indicate that ventilatory responses to hypoxia and hypercapnia and respiratory patterns while breathing air include genetically determined factors, whereas perception threshold for added resistance and respiratory patterns during hypercapnia are influenced predominantly by environmental forces in adulthood. (Auth. Abs. Mod.)

84–1417. Menkes, H. A.; Cohen, B. H.; Beaty, T. H.; Newill, C. A.; Khoury, M. J. **Risk Factors, Pulmonary Function, and Mortality.** In: *Genetic Epidemiology of Coronary Heart Disease: Past, Present, and Future.* Progress in Clinical and Biological Research, Volume 147. New York, Alan R. Liss, Inc., 1984, pp. 501-521.

Age and smoking were the most important of 11 risk factors found to be significantly associated with airways obstruction in a genetic—epidemiologic study of obstructive airways disease that investigated relationships among risk factors, pulmonary function, and mortality in 2,539 nonpatient adults. In cross-sectional evaluation, multiple factors were associated with airways obstruction. Of these, smoking appears to interact strongly with other factors. In longitudinal assessment, risk factors associated with an accelerated loss of function included gender, age, smoking, blood type, and initial closing capacity; however, these factors accounted for only a small percentage of the variance in loss of function over a 4.7-year followup period. Airways obstruction itself was an important independent risk factor for subsequent mortality, with most deaths not primarily attributable to the pulmonary system. Results indicate that pulmonary dysfunction may be important in the evolution of extrapulmonary diseases. Thus, both pulmonary function and associated constitutional factors should be evaluated when studying factors aimed at intervention. (Auth. Abs. Mod.)

84–1418. Milner, A. D. **Pediatric Lung Disease: Can Adult Sequelae Be Prevented?** *Journal of Respiratory Diseases* 5(4): 81-90, April 1984.

The most important step in the prevention of chronic obstructive pulmonary disease in adults is to discourage teenage smoking. There is strong evidence that acute and chronic lower respiratory tract diseases occurring in childhood cause damage that can develop into chronic obstructive pulmonary disease in adults. In particular, acute bronchiolitis, asthma, bronchiectasis, and bronchopulmonary dysplasia are linked to decreased adult lung function. Aggressive measles and pertussis immunization policies, and possibly breast feeding, are of some help in preventing pediatric respiratory infections, but the main aim in fighting chronic bronchitis and emphysema in adults should be to prevent smoking by adolescents. Since attempts to shock young children by showing horror pictures of the long-term effects of smoking have not been successful, strategies may need to be more subtle to overcome the strong advertising campaigns and peer pressures to smoke. Pregnant women and young parents should also be counseled not to smoke. (Auth. Abs. Mod.)

84–1419. Mok, J.Y.Q.; Simpson, H. **Symptoms, Atopy, and Bronchial Reactivity After Lower Respiratory Infection in Infancy.** *Archives of Disease in Childhood* 59(4): 299-305, April 1984.

The prevalence of subsequent respiratory symptoms and the relationship between atopic status and bronchial reactivity were studied in 200 index children and controls 7 years after acute lower respiratory tract infections in infancy. Index children with recurrent symptoms differed from controls in social and family characteristics and atopic background: both index subgroups came from lower social classes than controls, and index children without bronchial reactivity were more apt to have a father who smoked (81.1 percent, $p<0.05$) compared with controls (67.6 percent). Index subgroups with and without bronchial reactivity had increased cough and wheeziness compared with respective matched controls. The former included children with "established" asthma and the latter those with "established" bronchitis. Atopic backgrounds were similar in both subgroups, with no differences between cases and controls. Findings suggest that atopic background and bronchial reactivity are not closely related, but may contribute independently to the persistence of symptoms after respiratory infections in infancy. Bronchial reactivity may be a more useful basis than atopic status on which to separate children with episodic cough or wheeze, or both, into asthmatic and bronchitic subgroups. (Auth. Abs. Mod.)

84–1420. Oleru, U. G. **Pulmonary Function of Exposed and Control Workers in a Nigerian Nonsoapy Detergent Factory.** *Archives of Environmental Health* 39(2): 101-106, March-April 1984.

Among smokers, those not previously exposed to dusty jobs had higher forced expiratory volume in 1 sec (FEV_1) and forced vital capacity (FVC) than those previously exposed, while nonsmokers who had not been previously exposed had values closer to that predicted. The difference between smokers and nonsmokers was not statistically significant, which may be attributed to the small number of smokers (25.8 percent) and low consumption levels. Respiratory symptoms and spirometric pulmonary function data for 128 males exposed to alkyl benzene sulfonate in a detergent factory and for 56 unexposed workers are reported. Exposed subjects generally complained of cough and mucus secretions, nasal catarrh, chest pain, and breathlessness. Unexposed workers had significantly lower frequency of symptoms and significantly higher FEV_1 and FVC than exposed workers. The reduction in pulmonary function of exposed subjects from the predicted was significantly higher than that experienced by unexposed subjects. Respiratory symptoms in exposed subjects decreased with duration of employment, which probably indicates the exodus from the work force of those who could not tolerate the detergent. (Auth. Abs. Mod.)

84–1421. Palosuo, T.; Andersson, M.; Vaananen, R.; Pyhala, R.; Koskenvuo, M.; Aho, K. **Serum Acute-Phase Glycoproteins and Immune Complexes in Relation to Smoking and Respiratory Symptoms: An Epidemiological Study.** *European Journal of Respiratory Diseases* 65(3): 210-215, April 1984.

A significant number (38 percent) of middle-aged heavy smokers exhibited elevated values of serum acute-phase glycoproteins, measured as a function of smoking status in 346 young Finnish conscripts, 228 middle-aged males, and 158 adults with persistent respiratory symptoms. Subjects, classified as nonsmokers, light smokers, and heavy smokers, were measured using a nephelometric assay of concanavalin A binding. Findings in conscripts, regardless of smoking status, did not differ from those found in sera of 48 blood donors serving as normal controls. In subjects with persistent respiratory symptoms, corresponding values were even higher (52 percent) than in heavy smokers, but no differences were recorded between smokers and nonsmokers. Elevated amounts of circulating immune complexes, measured by conglutinin-binding assay, were detected in 6 percent of middle-aged males and in 20 percent of subjects with persistent respiratory symptoms. The accuracy of the concanavalin A binding assay, the narrow distribution of binding values in healthy subjects, and the marked differences in the occurrence of elevated levels observed emphasizes the value of the test in epidemiological surveys.

84–1422. Petersen, M.; Castellan, R. M. **Prevalence of Chest Symptoms in Nonexposed Blue-Collar Workers.** *Journal of Occupational Medicine* 26(5): 367-374, May 1984.

Prevalence estimates for various respiratory symptoms, by smoking status, are presented for a blue-collar population with minimal previous occupational exposure to hazardous substances. A standard respiratory questionnaire was administered to 1,372 workers at 14 plants in North Carolina, and linear logistic prediction equations were developed for several symptoms: cough, phlegm production, dyspnea, wheezing, and chest illness under a variety of circumstances. Analysis was performed separately for the 651 smokers, 207 ex-smokers, and 514 never smokers. The variables of sex, race, age, height, weight, and education were also taken into account. Results can be used as descriptive statistics, as prediction equations from an external control group, and as a guide for adjustment of symptom prevalences in other population studies. The symptom portion of the questionnaire is included, and examples of the practical application of the results are given.

84–1423. Pratt, P. C.; Lynn, W. S. **Cotton Dust, Smoking, and the Lung. (Letter).** *Annals of Internal Medicine* 100(4): 618-619, April 1984.

A response to a previous letter (*Annals of Internal Medicine* 100(1): 162, January 1984) concerning lung function among retired cotton workers emphasizes that retirees who are nonsmokers or ex-smokers should show improvement in lung function. Because active workers and retirees were analyzed together, the more rapid decline in the active workers implies an improving lung function in the retirees; the worsening among active workers would subtract from improving values among retirees. Also, since lung function values among smoking retirees and active workers were reduced at followup, nonsmoking and ex-smoking retirees should show an even more striking improvement. However, the original research does not show data separately by smoking status. See also 84-0779, 84-0791, 84-0792, 84-1408.

84–1424. Pratt, P. C.; Vollmer, R. T. **The Beneficial Effect of Alcohol Consumption on the Prevalence and Extent of Centrilobular Emphysema. A Retrospective Autopsy Analysis.** *Chest* 85(3): 372-377, March 1984.

Associations between age, smoking, and alcohol consumption in the development of emphysema, pulmonary pigmentation, and airway lesions are examined. Inflation-fixed autopsied lungs (n=204) were divided into normal (93), centrilobular emphysema (CLE, 88), and other chronic processes (23). A review of case histories revealed 173 subjects had been smokers. Logistic analysis showed alcohol use was significantly associated with reduced extent of CLE even after allowing for age and smoking effects. More than half (56.1 percent) of the nondrinking smokers showed at least trace levels of CLE. None of the 31 nonsmokers had as much as 20 percent emphysematous involvement. It is suggested that the apparent prophylactic effect of alcohol against CLE probably results from inhibition of inflammatory cells. A drug with similar effects, but without the deleterious side effects of alcohol, might provide a meaningful degree of protection against emphysema in smokers.

84–1425. Pride, N. B. **Which Smokers Develop Progressive Airflow Obstruction?** *European Journal of Respiratory Diseases* 64(Supplement 126): 79-83, 1983.

Although the relationship between smoking and progressive airway narrowing is well established, only 10 to 20 percent of smokers develop severe airway narrowing. It may be that pulmonary exposure to tobacco smoke differs considerably, but studies have failed to correlate consumption or tar levels with lung function. A stronger relationship is found between accelerated decline in lung function and nonspecific bronchial reactivity to inhaled histamine and methacholine. However, increased reactivity may be a consequence, and not a cause, of reduced airways. Evidence for an allergic, atopic factor is weak. Severe α_1-antitrypsin deficiency is a strong risk factor for obstructive airways disease, but the more common lesser defects of α_1-antitrypsin appear to play a surprisingly unimportant role. The hypothesis that those destined to develop obstructive airways are those with chronic cough and recurrent infections has not been confirmed. The loss in lung function observed with infections is transient, while the association between cough and reduced lung function appears to be due to the association of both with smoking. For intervention purposes, an initial forced expiratory volume in 1 sec more than two SD below average is the best predictor of an accelerated decline in lung function.

84–1426. Public Health Service. **Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, December 1983, 13 pp.

Data sets from four community studies (East Boston, Massachusetts; Tucson, Arizona; Tecumseh, Michigan; and the Six Cities Study) were analyzed in a workshop sponsored by the National Heart, Lung, and Blood Institute on the health effects of passive smoking. Only one of the studies was longitudinal (East Boston, 7 years), and none directly addressed passive smoking's effect on the respiratory system, although all provided a considerable amount of relevant data. It was concluded that the effect of passive smoking on the respiratory system varies from negligible to quite small (0 to 3 percent reduction in forced expiratory volume in 1 sec). It was not possible to determine whether there are specific groups at increased risk. It is recommended that new large-scale population studies not be initiated (with the exception of the 0- to 5-year-old age group) until existing data sets have been evaluated. However, there is an urgent need for development of noninvasive biological indicators of exposure.

84–1427. Radermecker, M.; Gustin, M.; Saint-Remy, P. **Lack of Correlation Between Serum Angiotensin-Converting En-**

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zyme Activity and Bronchoalveolar Lymphocytosis in Lung Sarcoidosis. *European Journal of Respiratory Diseases* 65(3): 189-195, April 1984.

Among patients with pulmonary sarcoidosis, data indicate that smoking has no significant effect on serum angiotensin-converting enzyme (SACE) levels, but significantly depresses the alveolar lymphocytic reaction. The relationship between SACE activity and the percentage of lymphocytes in bronchoalveolar lavage fluid (BALF) was studied in 23 patients, 20 recently diagnosed with Stage I or II sarcoidosis, and 3 in Stage III. SACE and BALF lymphocytosis were elevated in 80 and 85 percent, respectively, of the Stage I and II patients, but were not significantly correlated. Of the five patients with discordant values, three nonsmoking Stage I patients had elevated BALF lymphocytosis and normal SACE and two smokers had elevated SACE and normal BALF lymphocyte percentages. Both SACE and BALF lymphocytosis were normal in Stage III patients. It is concluded that smoking exerts a masking effect on the alveolar lymphocytic reaction associated with active sarcoidosis.

84–1428. Reynolds, H. Y.; Chretien, J. **Respiratory Tract Fluids: Analysis of Content and Contemporary Use in Understanding Lung Diseases.** *DM Disease-a-Month* 30(5): 1-103, February 1984.

Smoking-related changes in normal lung fluids are included in a review of the content and diagnostic value of respiratory tract secretions. Formation of fluids by the mucous membrane, the types of secretory cells, the capillary-alveolar interface, and conservation of fluid through absorption are covered. Sampling methods and analysis of various subdivisions of respiratory tract fluid (nasal, oropharyngeal, tracheobronchial, bronchoalveolar, and expectorant) are also discussed. Lung fluids of smokers are distinctive from those of nonsmokers, and differences do not immediately revert upon cessation. There are about 20 differences between the bronchoalveolar lavage components of smokers and nonsmokers, the most obvious being cellular recovery. Smokers' lavage may contain four to five times more total cells, with higher proportions of alveolar macrophages and polymorphonuclear cells, but fewer lymphocytes. In addition, smokers' macrophages show changes in metabolic rate, ultrastructure, function, replication index, and response to chemotactic stimuli. Characteristics of fluid from patients with asthma, adult respiratory distress syndrome, alveolar proteinosis, acquired immunodeficiency disease of the lung, and interstitial and inflammatory lung disease are included.

84–1429. Ruckley, V. A.; Gauld, S. J.; Chapman, J. S.; Davis, J.M.G.; Douglas, A. N.; Fernie, J. M.; Jacobsen, M.; Lamb, D. **Emphysema and Dust Exposure in a Group of Coal Workers.** *American Review of Respiratory Disease* 129(4): 528-532, April 1984.

Emphysema occurs more frequently in coal miners who are smokers (72 percent) or ex-smokers (65 percent) than in nonsmoking coal miners (42 percent), according to post-mortem analysis of 450 miners who had been studied previously in a long-term epidemiologic project at 24 British mines. This average gradient was less marked in those with massive fibrosis (PMF) than in those with small pneumoconiotic lesions. In cases of PMF, 75 percent of nonsmokers had emphysema compared with 77 percent of ex-smokers and 85 percent of current smokers. Emphysema was associated with increasing evidence of pneumoconiotic disease in all smoking categories, and both panacinar and centriacinar emphysema occurred more frequently in smokers than in nonsmokers. Results confirm the expected association between prevalence of emphysema and age/smoking habit.

84–1430. Russell, M.A.H. **Smoking, Nicotine Addiction and Lung Disease.** *European Journal of Respiratory Diseases* 64(Supplement 126): 85-89, 1983.

Preventive measures to reduce smoking are the most important part of any strategy to reduce premature death and disability from lung cancer, chronic bronchitis, and many other diseases. Previous failures to deal more successfully with the smoking problem stem mainly from a failure to recognize it as a predominantly drug-taking activity. At the individual level, availability of a new form of treatment (nicotine chewing gum) has greatly enhanced success rates. Generally, switching to a pipe, cigars, or low-yield cigarettes does not lead to the expected reduction in the intake of harmful smoke components and is, consequently, unlikely to be beneficial. Snuff is the most promising approach. If smokers in Great Britain were to switch from cigarettes to snuff, there would be 55,000 fewer deaths each year from lung cancer and chronic bronchitis. History suggests that a switch to snuff is more feasible than elimination of smoking. It would also be more humane, since, unlike smoking, it would not be a source of annoyance and harm to nonusers. (Auth. Abs.)

84–1431. Schachter, E. N.; Doyle, C. A.; Beck, G. J. **A Prospective Study of Asthma in a Rural Community.** *Chest* 85(5): 623-630, May 1984.

Asthmatics were as likely to smoke as nonasthmatics among 1,303 white residents of a rural community (Lebanon, Connecticut) examined in 1972 and again in 1978. A total of 73 asthmatics and 278 persons who complained of wheezing were identified in 1972. At followup, 68 percent of the asthmatics were in remission, and 77 percent of those with wheeze had improved. Both smoking and chronic bronchitis were associated with wheeze in all subjects, and chronic bronchitis was associated with asthma in women over 18 years of age. Also, loss of pulmonary function over time was consistently greater for asthmatic adults, with a trend toward poorer function observed in subjects with chronic asthma. It is concluded that (1) remission rates among asthmatics aged above 7 years are high, and (2) asthma is associated with loss in pulmonary function, but (3) asthmatics are as likely to smoke as nonasthmatics.

84–1432. Sixt, R.; Bake, B.; Oxhoj, H. **The Single-Breath N_2-Test and Spirometry in Healthy Non-Smoking Males.** *European Journal of Respiratory Diseases* 65(4): 296-304, May 1984.

Comparison of asymptomatic smokers with asymptomatic nonsmokers supports the conclusion that smokers should not be included in groups used for establishing prediction formulae. Even the asymptomatic smoker tends to have a lower forced expiratory volume in 1 sec (FEV_1) and higher slope of the alveolar plateau (ΔN_2) than the nonsmoker without symptoms. Data are from a study of a random population sample of 178 healthy males that excluded all smokers, to establish reference equations for variables from the N_2-test and for ordinary spirometry. Using the single-breath N_2-washout test, closing volume (CV) and ΔN_2 were measured. A prediction equation for CV was produced by simple regression on age. ΔN_2 showed an accelerated increase with age above the age of 50. A curvilinear equation was established, as were reference equations for vital capacity (VC), FEV_1, and FEV_1 in percent VC (FEV percent) by means of linear multiple regression on age and height. The distribution of ΔN_2 was not homogenous throughout the age range. As an upper limit of the reference range, 180 percent of the predicted value is suggested. (Auth. Abs. Mod.)

84–1433. Thelin, A.; Tegler, O.; Rylander, R. **Lung Reactions During Poultry Handling Related to Dust and Bacterial Endotoxin Levels.** *European Journal of Respiratory Diseases* 65(4): 266-271, May 1984.

In 47 poultry workers, no significant differences were found in symptoms reported by smokers (n=20) and nonsmokers (n=27). Airborne dust and endotoxin levels on poultry farms were determined for various working processes known to involve heavy exposure. Workers at different sites were studied by questionnaires for work-related symptoms. Lung function measurements were made before and after work. Dust levels exceeded the standard for organic dust, and endotoxin levels exceeded those known to cause respiratory and other symptoms. The average decrease in forced expiratory volume in 1 sec over the working day(s) ranged from 0.07 to 0.19 liters. Upper airway irritation was present in one-third of the workers, and about 10 percent complained of chest tightness. The effect may have been due to the high amounts of dust causing a general respiratory irritation and to the endotoxin. (Auth. Abs. Mod.)

84–1434. Voisin, C.; Aerts, C.; Fournier, E.; Guiselin, M.; Marquillies, P.; Merdy, C.; Jacob, A.; Fourneau, C. **Alveolar Macrophages Versus Toxic Gases. A Controlled In Vitro Approach.** In: Favez, G.; Junod, A.; Levenberger, P. (Editors). *The Cells of the Alveolar Unit.* Current Problems in Clinical Biochemistry, No. 13. Vienna, Huber Publishers, 1982, pp. 212-222.

Guinea pig alveolar macrophages showed significant cytotoxicity after short-term exposure to the gas phase of cigarette smoke. Macrophages were layered on a Gelman membrane and applied to the surface of a reservoir of culture medium (gaseous phase culture). Cells exposed to smoke from 3 to 5 cigarettes, passed through a Cambridge filter (35 ml puff/min) showed morphologic alterations of cytoplasmic and nuclear structures, a decrease in bactericidal activity, and reduced ATP content. Large variations in toxicity were observed, depending on cigarette brand, and appeared to be related to nitrogen dioxide (NO_2) levels. Long-term (24 hr) exposure to NO_2 alone produced significantly negative effects on the antioxidant enzyme properties of the macrophages: 30-min exposure to 2 and 5 ppm NO_2 completely destroyed the cell preparation. In addition, a 30-min exposure to acrolein alone (4 to 35 ppm) resulted in a significant decrease in ATP levels. The effects of ozone were also determined.

84–1435. Voisin, C.; Fournier, E. **Alveolar Macrophages in Pulmonary Defence.** *European Journal of Respiratory Diseases* 64(Supplement 126): 35-38, 1983.

Inhaled pollutants do not kill alveolar macrophages (AM), but induce a disturbance of their secretory activities; e.g., in smokers, prolonged morphological and functional disturbances are observed. Phagocytic index and bactericidal ability remain normal, but NCF, elastase, and superoxide anions secretion contribute to explain the protease—antiprotease imbalance, leading to emphysematous lesions, especially in patients with α_1-antitrypsin deficiency. In addition, immunological dysfunctions have been described: decrease in immunoglobin G membrane receptors and in migration activity, and probably disturbances in regulation of lymphocyte function. There is also an enzymatic adaptation (an increase in aryl hydrocarbon hydroxylase) found in smokers that is capable of potentiating AM activities. Other AM activities (phagocytosis, role in immunology, and secretory functions) are described, and factors influencing AM efficiency are discussed. While AM are not the only cells that intervene in the defense of the respiratory tract against inhaled pollutants or microorganisms, they appear to be the first defenders of the lung under attack.

84–1436. Warner, M. A.; Divertie, M. B.; Tinker, J. H. **Preoperative Cessation of Smoking and Pulmonary Complications in Coronary Artery Bypass Patients.** *Anesthesiology* 60(4): 380-383, April 1984.

The respiratory complication rate in 456 smokers undergoing coronary bypass surgery improved dramatically among those who were abstinent for >8 weeks prior to surgery. Of 500 patients surveyed retrospectively, pulmonary complication incidence among those who quit >8 weeks before surgery (n=176) was between 15 and 20 percent, depending on time elapsed, compared with nonsmokers (n=44), who had an 11.4

percent complication rate. Among those who continued smoking ($n=124$) and those who quit <8 weeks prior to surgery ($n=156$), rates were 48.4 percent and 56.4 percent, respectively. Cessation of smoking >4 weeks before surgery decreased the incidence of productive cough, but did not influence complication rates. Preoperative impaired pulmonary function showed no influence on incidence of postsurgical respiratory problems.

84–1437. Yeung, M. C.; Dy Buncio, A. **Leukocyte Count, Smoking, and Lung Function.** *American Journal of Medicine* 76(1): 31-37, January 1984.

Leukocyte counts were significantly higher among current smokers compared with nonsmokers and ex-smokers in 1,826 white male workers (776 smokers, 652 ex-smokers, 398 nonsmokers) in a pulp and paper mill and 1,620 white male workers (774 smokers, 509 ex-smokers, 337 nonsmokers) in an aluminum smelter in British Columbia. The relationship between leukocyte count, cigarette smoking, and pulmonary function was examined in workers taking part in epidemiologic health studies that included a medical and occupational questionnaire, spirometric measurements, and leukocyte count. Air contaminants in the work environment were also measured. No significant increase in the prevalence of respiratory symptoms and lung function abnormalities was found between the exposed and nonexposed workers in each work population. Similarly, no difference in leukocyte count was found between exposed and nonexposed workers. In both populations, there was an inverse correlation between leukocyte count and forced expiratory volume in 1 sec and forced vital capacity of the worker, regardless of smoking status, suggesting that the leukocyte response to external stimuli may be another determinant of lung function measurements. (Auth. Abs. Mod.)

84–1438. Zamel, N.; Webster, P. M. **Improved Expiratory Airflow Dynamics With Smoking Cessation.** *Clinical Respiratory Physiology* 20(1): 19-23, 1984.

The relationship between maximum expiratory flow (Vmax) and lung recoil (Pst(1)) breathing air or a mixture of 80 percent helium plus 20 percent oxygen (HeO_2) was examined in five male and five female subjects (33.4±6.4 years) before and after 2 months' abstinence from smoking. Pst(1) dependence of Vmax, a relationship dependent on airway elastance at the flow-limiting mechanism, or "choke," was estimated by measuring the slope and the zero flow intercept using the maximum flow-static recoil (MFSR) curve. MFSR slope was unchanged by abstinence. The intercept showed no difference between air and HeO_2 before or after smoking cessation. Density independence of the zero flow intercept can be assumed from wave speed theory if there is no change at the "choke" and the slope truly reflects the elastic behavior of the "choke." This correspondence of theory and measurement suggests that the "choke" was not changed by abstinence. The reduction of Pst(1) with smoking cessation, without any fall in Vmax at the same lung volume, suggests a complementary reduction in lung recoil and frictional energy loss upstream of the "choke." This suggests that a decrease in small airway muscle tone was the single cause of a reduction of Pst(1) and peripheral airway resistance. (Auth. Abs.)

CARDIOVASCULAR DISEASES

84–1439. Aberg, A.; Bergstrand, R.; Johansson, S.; Ulvenstam, G.; Vedin, A.; Wedel, H.; Wilhelmsson, C.; Wilhelmsen, L. **Declining Trend in Mortality After Myocardial Infarction.** *British Heart Journal* 51(3): 346-351, March 1984.

Two-year post-myocardial infarct mortality among 1,322 patients in Goteborg, Sweden, dropped from 14.5 to 6.4 percent between 1968 and 1977, a period when the number of smokers who quit increased from 47 to 65 percent ($p=0.025$) and the patients treated with beta blockers increased from 4 to 70 percent ($p<0.0001$). The proportion of smokers at the time of infarct decreased from 92 to 72 percent ($p=0.018$), and angina pectoris prevalence decreased from nearly 50 to just over 30 percent ($p=0.0006$). The higher tendency among smokers to quit and lower prevalence of angina pectoris could explain only part of the mortality reduction; the majority is probably due to treatment with beta blockers.

84–1440. Agner, E.; Morck, H. I. **The Significance of Systolic Hypertension in the 1897-Population in Glostrup.** *Acta Medica Scandinavica* Supplement 676: 15-21, 1983.

Results of a 10-year prospective study of risk markers for cardiovascular events showed that neither high diastolic blood pressure (BP) or high systolic BP had individual predictive value for death, but high systolic BP had individual predictive value in both sexes for development of cardiovascular disease. In 1967, among the sample of 70-year-old adults (230 men, 210 women), 24 percent of the men and 8 percent of the women were smokers who inhaled. In men, this was related to low systolic and low diastolic BP. At age 70, mean systolic BP was 147 and mean diastolic BP 82 in men who inhaled, compared with 159 and 87, respectively, in nonsmoking men (significant at the 1 percent level). Mean systolic BP in women who inhaled was 158 and mean diastolic BP 82, compared with 159 and 82, respectively, in nonsmoking women. Among women, only the systolic BP correlated significantly with smoking. Overall findings suggest that high arterial BP in old age is still important as a risk marker for development of cardiovascular disease, although to a lesser degree than has been shown in studies of younger populations.

84–1441. American Family Physician. **Cigarette Smoking Is Leading Modifiable Cause of CHD.** *American Family Physician* 29(2): 331, 333, February 1984.

Cigarette smoking is both a major cause of and the most important modifiable risk factor for coronary heart disease (CHD). Approximately 30 percent of CHD deaths are attributable to smoking. Cigarette users have two to four times the risk for CHD compared with nonsmokers, and the risk appears to be dose-related. When high serum cholesterol and hypertension are also present, the risk is eight times higher. Smoking is also a significant factor in the etiology of peripheral vascular disease. The associations are confirmed by smoking cessation. Data show a substantial reduction in risk for CHD within a few years of quitting, and after 10 years, the risk is almost equivalent to that of nonsmokers. The antismoking movement has been successful in that the percentage of adults who smoke dropped from 43 in 1966 to 33 in 1980, and an estimated 200,000 premature deaths were avoided between 1964 and 1978. However, more than 300,000 smoking-related CHD deaths still occur each year.

84–1442. Barrett-Connor, E.; Khaw, K. **Family History of Heart Attack as an Independent Predictor of Death Due to Cardiovascular Disease.** *Circulation* 69(6): 1065-1069, June 1984.

Data from a 9-year followup of 1,774 men and 2,240 women who had reported no known cardiovascular disease showed that younger women with a positive family history were more likely to smoke cigarettes. Among those aged 40 to 59, 50 percent of the women with a family history of heart attack before 50 years of age were smokers compared with only 35.1 percent of men with a similar history. The percentage of women smokers was higher for all categories except men aged 60 to 79 with a family history of heart attack before age 50 (21.1 and 21.4 percent, respectively). The independent contribution of a positive family history of heart attack to subsequent cardiovascular death was determined by the Cox model after adjusting for age, systolic blood pressure, total plasma cholesterol level, obesity, cigarette smoking, personal history of diabetes, and estrogen use (in women). In men, but not in women, a positive family history of heart attack was independently predictive of death from all causes and from cardiovascular and ischemic heart disease. Significant differences were restricted to younger men; those with a positive family history had a fivefold excess risk of cardiovascular death independent of other risk factors. A family history of premature heart attack was not predictive of mortality in men or women. Differences between younger and older men probably reflect survivorship, but differences between men and women are unexplained. (Auth. Abs. Mod.)

84–1443. Belch, J.J.F.; McArdle, B. M.; Burns, P.; Lowe, G.D.O.; Forbes, C. D. **The Effects of Acute Smoking on Platelet Behaviour, Fibrinolysis and Haemorheology in Habitual Smokers.** *Thrombosis and Haemostasis* 51(1): 6-8, February 28, 1984.

An increased tendency to hypercoagulability, which became more pronounced immediately after smoking three cigarettes, was found in smokers compared with controls. The effects of acute smoking on platelet behavior, fibrinolysis, and hemorrheology were studied in 10 habitual smokers (7 females, 3 males) and compared with age- and sex-matched nonsmoking controls. Subjects had smoked between 15 and 20 cigarettes/day for at least 4 years, but none had smoked for at least 10 hours before testing. Blood was sampled prior to the first cigarette and 10 minutes after the third cigarette was smoked. Results show that smokers had higher plasma fibrinogen ($p<0.04$), lower plasminogen ($p<0.02$) and plasminogen activator ($p<0.05$), and higher plasma viscosity ($p<0.003$). Changes recorded after smoking the three cigarettes were an increase in rate of platelet aggregation to ADP ($p<0.02$), and an increase in α_2 macroglobulin ($p<0.02$) and factor VIII RAG ($p<0.05$). Plasma viscosity was decreased ($p<0.02$), as was red cell deformability ($p<0.02$). (Auth. Abs. Mod.)

84–1444. Candelise, L.; Bianchi, F.; Galligoni, F.; Albanese, V.; Bonelli, G.; Bozzao, L.; Inzitari, D.; Mariani, F.; Rasura, M.; Rognoni, F.; Sangiovanni, G.; Fieschi, C. **Italian Multicenter Study on Reversible Cerebral Ischemic Attacks: III—Influence of Age and Risk Factors on Cerebrovascular Atherosclerosis.** *Stroke* 15(2): 379-382, March-April 1984.

Evidence indicating a possible causal relationship between smoking and amount of atherosclerosis was found in a study of the influence of smoking, age, hypertension, diabetes, and dyslipidemia in 462 patients with reversible ischemic attacks who had cerebral angiography. The degree of atherosclerosis was quantified using extracranial and intracranial cerebrovascular scores (ECS, ICS) based on the number and severity of the lesions in 11 extracranial and 21 intracranial arterial segments. Cigarette smoking (53 percent of patients smoked >10 cigarettes/day) was the only risk factor that showed a strong association with the extracranial score independent of the effect of age and other risk factors. Of the patients under age 45, 36 percent had a normal angiogram compared with 17 percent of patients over 45 years of age. In the subgroup of patients with abnormal angiogram, the mean ECS and ICS vascular scores were not significantly different in the two age groups. (Auth. Abs. Mod.)

84–1445. Carleton, R. A.; Lasater, T. M. **Coronary Heart Disease and Human Behavior.** *Preventive Medicine* 12(5): 610-618, September 1983.

Cigarette smoking, high cholesterol levels, high blood pressure, obesity, and poor physical fitness are all major risk factors contributing to coronary heart disease resulting from atherosclerosis, and most are greatly influenced by human behavior. An approximate dose—response relationship has been established between smoking and the risk of coronary heart disease, and smoking has been identified as a risk factor for sudden, unanticipated death. Since smoking also plays a role in the development of diverse forms of cancer and chronic pulmonary disease, it should be one of the major targets for any public health effort. While it is still unclear exactly how smoking produces such adverse health effects, suggested causes include activation of the adrenergic system and the effects of nicotine and carbon monoxide. Because smoking is heavily influenced by social pressures, health professionals are uniquely situated to affect this behavior through recommendations and actions. Guidelines are presented on effective role models for physicians, and techniques are described for promoting positive smoking decisions by individual patients. Potential interactions among the several behavioral health factors and the opportunities to capitalize on motivation for change are emphasized, suggesting that modification of several health factors could be attempted simultaneously.

84–1446. Cronenwett, J. L.; Warner, K. G.; Zelenock, G. B.; Whitehouse, W. M., Jr.; Graham, L. M.; Lindenauer, S. M.; Stanley, J. C. **Intermittent Claudication: Current Results of Nonoperative Management.** *Archives of Surgery* 119(4): 430-436, April 1984.

Cigarette smoking was examined as one high risk factor for clinical deterioration and subsequent operative intervention in 91 patients with mild intermittent claudication who were followed for 6 to 72 months. Smoking incidence, assessed before, during, and at the end of the study, showed that 96 percent had smoked cigarettes for 60±33 pack-years (range, 10 to 160) and 68 percent had smoked at least 40 pack-years. At onset, 4 percent were never smokers, 17 percent ex-smokers, and 70 percent current smokers. During the study, 31 percent stopped smoking, 26 percent reduced consumption by at least 50 percent, and 43 percent continued smoking at the same frequency. At followup examination, patients who smoked at onset had decreased consumption from 1.54±0.68 packs/day to 0.76±0.54 packs/day, a 51 percent reduction. However, there were three times as many operations among patients who had smoked ≥40 pack-years as among patients who had smoked <40 pack-years. Patients who stopped or decreased smoking had slightly less symptom progression. Major daily exercise was associated with stable claudication. Careful followup of patients exhibiting this early symptom of lower extremity arterial occlusive disease is recommended to allow timely operative intervention when required.

84–1447. Deanfield, J.; Wright, C.; Krikler, S.; Ribeiro, P.; Fox, K. **Cigarette Smoking and the Treatment of Angina With Propranolol, Atenolol, and Nifedipine.** *New England Journal of Medicine* 310(15): 951-954, April 12, 1984.

Data show that smoking has direct and adverse effects on the heart and interferes with the efficacy of antianginal drugs, particularly nifedipine. Ten cigarette smokers with angina received placebo, nifedipine (60 mg/day), propranolol (240 mg/day), and atenolol (100 mg/day) to determine if smoking affects the results of drug treatment. The 4-week, double-blind procedure was repeated with the same randomly determined order of drug sequences after all 10 subjects had stopped smoking. Before and after quitting, all three drugs significantly reduced the frequency of angina, as measured with angina diaries, and improved the results of maximal exercise testing and 48-hour ambulatory monitoring of ST segments. During the nonsmoking phase, however, there was an overall decline in frequency of angina and improved performance on exercise testing, compared with the smoking period. Results of 48-hour ambulatory monitoring remained unchanged. With nifedipine, there was more improvement after smoking cessation than with the other two drugs or placebo. Blood levels of propranolol were increased when patients stopped smoking; levels of nifedipine and atenolol were unchanged. (Auth. Abs. Mod.)

84–1448. Debert Ribeiro, M. B. **Hypertension Among Female Workers in Sao Paulo, Brazil. Predictors and Joint Effects.** *Hypertension* 5(6, Part III): V-144-V-148, November-December 1983.

Diastolic blood pressure (DBP) was lowest (mean, 66.1 mm Hg) among very young, lean women who smoked in an investigation of predictive factors for hypertension among women in Brazil. Six independent variables emerged after analysis using the Morgan-Sonquist procedure on interactive effects: smoking, age, Quetelet index, occupation, education, and socioeconomic level. Older women in production-linked occupations had the highest DBP (mean, 89.5 mm Hg), and young, overweight women without a college education had intermediate levels (mean, 71.5 mm Hg). It is noted that although the difference in DBP due to smoking appears small (3.4 mm Hg), it cannot be accounted for by weight since it was detected only among lean women.

84–1449. Ellis, N.; Lloyd, B.; Lloyd, R. S.; Clayton, B. E. **Selenium and Vitamin E in Relation to Risk Factors for Coronary Heart Disease.** *Journal of Clinical Pathology* 37(2): 200-206, February 1984.

Fasting blood samples from 116 volunteers (66 nonsmokers, 41 cigarette smokers, 9 pipe or cigar smokers) revealed significantly decreased glutathione peroxidase ($p<0.05$) and selenium ($p<0.001$) levels and significantly increased cadmium levels ($p<0.001$) in cigarette smokers compared with nonsmokers. The selenium:cadmium ratio was correspondingly lower in cigarette smokers. Multivariate analysis showed that the decrease in blood and serum concentrations of selenium and the increase in whole blood cadmium concentrations in cigarette smokers was independent of alcohol consumption. There was no correlation between blood selenium concentrations or glutathione peroxidase activities and cardiovascular disease risk factors. Neither alcohol consumption nor smoking had an effect on vitamin E concentrations. A strong association was found between vitamin E and serum lipid concentrations, although the increase in triglyceride concentrations in smokers was not matched by a comparable increase in vitamin E. The possible role of selenium in the etiology of heart disease remains unresolved.

84–1450. Fowler, A. W.; Cox, J. **Sudden Death in Squash Players. (Letters).** *Lancet* 1(8373): 393-394, February 18, 1984.

A relationship between sudden death from coronary heart disease (CHD) and smoking in squash players is suggested (although it had not been discussed as a factor in a previous study), since 15 of the 23 reported deaths occurred in men who had smoked >10 cigarettes/day (more than twice the frequency of smoking in the general population). Such active sports, often taken up by unfit, middle-aged men, cause the release of large quantities of catecholamines, as does smoking, leading to changes in heart rate and blood pressure and risk of arrhythmia and other factors associated with cardiac hypoxia. The value of screening squash players for risk factors is also discussed, noting that with over 1.5 million participants it would be an enormous task with small return. However, a short medical review of new players (especially those over 40 years of age) might help detect potential sudden-death victims.

84–1451. Grunwald, J.; Mey, J.; Schonleben, W.; Hauss, J.; Hauss, W. H. **Cultivated Human Arterial Smooth Muscle Cells. The Effect of Donor Age, Blood Pressure, Diabetes and Smoking on In Vitro Cell Growth.** *Pathologie Biologie* 31(10): 819-823, December 1983.

Human arterial smooth muscle cells cultured from smokers appear to have decreased proliferation capacity in vitro. Cultures from omental arteries of 153 patients were divided according to doubling time into fast-proliferating (FP) and slow-proliferating (SP) categories and compared for donor smoking status, age, blood pressure, diabetes mellitus, and carcinoma. Significantly more smokers' cells were of the SP type (9.9 percent compared with 7.7 percent FP). Proliferation rate was inversely proportional to age. Donors with high diastolic blood pressure and those with carcinoma also had significantly more SP cells, an indication of lower growth capacity. As a consequence, the cells reach their limited life span earlier, accounting for the higher proportion of senescent, slowly proliferating cells found in vitro. Results may provide relevant information on the pathogenesis of arteriosclerosis in man.

84–1452. Halawa, B. **Wplyw Jednorazowego Palenia Tytoniu na Krazenie Centralne, Stezenie Adrenaliny i Noradrenaliny w Surowicy Krwi i Karboksyhemoglobiny we Krwi. [Effect of One Dose of Cigarette Smoke on the Central Circulation, Serum Adrenaline and Noradrenaline Concentrations and Blood Carboxyhaemoglobin Concentration.]** *Wiadomosci Lekarskie* 36(20): 1653-1659, October 15, 1983, Polish.

Using radiocardiography, the effect of smoking two cigarettes on the central circulation, serum adrenalin and noradrenalin levels, and blood carboxyhemoglobin concentration was studied in healthy subjects and patients with stable angina. In healthy subjects, smoking two cigarettes in rapid succession caused a rise in cardiac index and left ventricular ejection index, increased heart rate, shortened postexercise time of pulmonary circulation, and decreased index of pulmonary blood volume. In patients with stable angina of effort, smoking two cigarettes increased only cardiac index and heart rate (determined at rest and after exercise). Consumption of two cigarettes increased adrenalin levels by 46 percent, noradrenalin levels by 20 percent, and carboxyhemoglobin concentration fourfold. (Auth. Abs.)

84–1453. Hart, J. T. **Still Nobody's Business? Prevention of Coronary Disease Through Primary Care.** *Practitioner* 228: 41-42, 45, 47, 49-50, January 1984.

Smoking is a reversible risk factor for coronary disease, especially for those under age 50, yet quitting is difficult and demands an enormous effort by the primary care team. Coronary heart disease mortality is not declining in Great Britain as it is in Australia and the United States, and the need to reorient primary care medicine from a preoccupation with health breakdown to the wider problems of health maintenance is emphasized. Patients who smoke do not require long-term medication with a substantial risk of side effects, as do those with hypertension. Thus, in all but hypertensive emergencies, smoking cessation should have a higher priority than blood pressure control. Even so, only a minority of hypertensives are under treatment and followup, and it is recommended that hypertension be treated only as a risk factor, not a disease. Smoking and obesity should also be treated as risk factors, rather than nonmedical problems. For primary care to reach its real preventive potential, the medical community must begin to treat causes rather than effects. The problems involved in reorientation toward preventive medicine are discussed.

84–1454. Hickey, N.; Mulcahy, R.; Daly, L.; Graham, I. **Mortality From Coronary Heart Disease—Trends for the Republic of Ireland.** *Postgraduate Medical Journal* 60(699): 9-14, January 1984.

A concomitant decrease in smoking prevalence and mortality from all causes among men in Ireland has been observed. Between 1972-1973 and 1978-1979, cigarette smoking prevalence declined from 43 to 36 percent, more in men (9 percent) than in women (6 percent). Among patients with first myocardial infarction (663 men <60 years old), 87.1 percent were smokers in 1967-1971 and only 66.1 percent in 1972-1975. It is concluded that these trends may be related, and that improved identification and treatment of hypertension may also have contributed to the recent decline in coronary heart disease mortality.

84–1455. Johansson, S.; Bergstrand, R.; Ulvenstam, G.; Vedin, A.; Wilhelmsson, C.; Wedel, H.; Wilhelmsen, L.; Aberg, A. **Sex Differences in Preinfarction Characteristics and Long-Term Survival Among Patients With Myocardial Infarction.** *American Journal of Epidemiology* 119(4): 610-623, April 1984.

Cigarette smoking was the only primary risk factor that showed a consistently higher prevalence in male patients than in female patients, and may contribute to the male preponderance in the incidence of myocardial infarction. The prevalence of primary risk factors, previous medical history, and physical activity were assessed among 262 women and 1,259 men who suffered a first nonfatal myocardial infarction (MI) between 1968 and 1977 in Goteborg, Sweden. During the first 5-year period, 64 percent of the women and 87 percent of the men were smokers, compared with 58 (not significant) and 77 percent ($p<0.001$), respectively, in the second 5-year period. Approximately 29 percent of the women and 49 percent of the men consumed >15 g tobacco/day. Women with infarctions had higher serum cholesterol levels ($p<0.001$) and higher blood pressure values ($p<0.001$), but were less apt to be smokers. Female patients also reported chest pain and dyspnea on exertion and low physical activity both at work and during leisure time significantly more often than men, differences that remained after controlling for estimated primary risk. Although more women below 45 years of age had hypertension and diabetes prior to MI, no similar and consistent differences were found between women and men in older age groups. The cumulative 5-year survival rate was 80 percent in women and 81 percent in men. Below age 45, the survival rate was lower among women than men ($p<0.01$). No sex difference was found in the recurrence rate of nonfatal reinfarctions. (Auth. Abs. Mod.)

84–1456. Keys, A.; Menotti, A.; Aravanis, C.; Blackburn, H.; Djordevic, B. S.; Buzina, R.; Dontas, A. S.; Fidanza, F.; Karvonen, M. J.; Kimura, N.; Mohacek, I.; Nedeljkovic, S.; Puddu, V.; Punsar, S.; Taylor, H. L.; Conti, S.; Kromhout, D.; Toshima, H. **The Seven Countries Study: 2,289 Deaths in 15 Years.** *Preventive Medicine* 13(2): 141-154, March 1984.

Analysis of time trends in relationships of mortality to entry characteristics showed the continued importance of smoking, age, and blood pressure, with a tendency for the importance of cholesterol to decline in the last 5 years of followup. Among 11,579 men aged 40 to 59 without evidence of cardiovascular disease, 2,289 died in 15 years, 618 from coronary heart disease. The 15 cohorts in seven countries (four regions) differed in all-causes death rate, mainly reflecting differences in coronary mortality. Among characteristics at entry, only mean blood pressure helped to explain cohort differences in all-causes death rate. Three-quarters of the variance in coronary death rate was accounted for by differences in mean serum cholesterol and blood pressure. Examination of mortality risk for individuals showed that (1) for coronary death, smoking, age, serum cholesterol, and blood pressure were highly significant in all regions except Japan, where there were too few coronary deaths for evaluation; (2) physical activity was significant only in southern Europe, where differences are associated with socioeconomic status; (3) and for all-causes death, smoking, age, and blood pressure were highly significant risk factors, except in Japan. Relative body weight tended to be a negative risk factor everywhere, significantly so in southern Europe. Expectations for coronary death from the experience in the United States and northern Europe greatly exceeded observed deaths in southern Europe for men of their age, serum cholesterol, blood pressure, smoking habits, physical activity, and relative weight. The reverse, prediction of coronary deaths in America and in northern Europe from the southern European experience, greatly underestimated the deaths observed. (Auth. Abs. Mod.)

84–1457. Klein, L. W.; Ambrose, J.; Pichard, A.; Holt, J.; Gorlin, R.; Teichholz, L. E. **Acute Coronary Hemodynamic Response to Cigarette Smoking in Patients With Coronary Artery Disease.** *Journal of the American College of Cardiology* 3(4): 879-886, April 1984.

Smoking increased coronary resistance in patients with coronary artery disease, and had a greater impact in patients with a severe proximal stenosis than in those with a distal stenosis. Coronary sinus blood flow after cigarette smoking was measured in 16 patients with coronary artery disease (group I) and in 6 patients without angiographically detectable coronary disease (group II). Seven patients (group IA) had severe (≥75 percent) proximal left coronary lesions and nine patients (group IB) had significant distal lesions with 50 percent or less proximal stenoses. All patients were either present or past chronic cigarette smokers. Group I had a smaller overall increase (1.6±5.3 percent) in coronary sinus blood flow than group II (7.7±6.1 percent, $p<0.05$). Coronary resistance increased overall (2.7±5.3 percent) in group I but decreased (2.4±3.4 percent) in group II ($p<0.05$). Patients in group IA had a highly significant increase in coronary resistance compared with group IB (7.0±4.2 percent vs. 0.9±2.6 percent, $p<0.001$). Coronary sinus flow tended to decrease (1.2±4.6 percent) in group IA but to increase (3.8±5.1 percent) in group IB ($p=0.06$). It is proposed that smoking increases coronary artery tone at the site of the stenosis, limiting the coronary flow response proportionally to the size of the affected vascular bed. (Auth. Abs. Mod.)

84–1458. Korner, P. I.; Bauer, G. E.; Doyle, A. E.; Edmondson, K. W.; Hodge, R. L.; Hunyor, S.; Hurley, T. H.; Leighton, P. W.; De Souza, D.; Lovell, R.R.H.; McCall, M. G.; McPhie, J. M.; Rand, M. J.; Reader, R.; Whyte, H. M.; Abernethy, J. D. **Prognostic Factors in the Treatment of Mild Hypertension.** *Circulation* 69(4): 668-676, April 1984.

In the Australian therapeutic trial of 3,427 subjects with mild diastolic hypertension, univariate analysis of covariates at entry that might be associated with prognosis showed a higher incidence of events leading to trial termination (trial end points) in cigarette smokers, older subjects, men, and those with higher systolic blood pressure and lower body mass index than in complementary groups. Trial end points were mostly due to ischemic heart disease (158 cases, 45 myocardial infarct) and cerebrovascular disease (37 events). There was a lower incidence in actively treated subjects (1,085 males, 297 smokers; 636 females, 121 smokers) than in untreated control subjects (1,085 males, 301 smokers; 621 females, 138 smokers) at both high and low levels of incidence of each covariate considered. Treatment benefit was most marked in those with lower serum cholesterol levels and lower systolic blood pressures. Multivariate regression analyses confirmed that the incidence of trial end points increased with age and with systolic blood pressure and showed higher rates in cigarette smokers, which increased markedly with decreasing body weight. Multivariate analysis of effects of treatment in smokers showed an increasing benefit with decreasing body weight. The apparent relationship of treatment benefit to systolic blood pressure and serum cholesterol level in the univariate analysis did not reach statistical significance in the multivariate analysis. Similar results were found when ischemic heart disease events alone were considered. (Auth. Abs. Mod.)

84–1459. Kuller, L. **Risk Factor Reduction in Coronary Heart Disease.** *Modern Concepts of Cardiovascular Disease* 53(2): 7-11, February 1984.

Cardiovascular and cerebrovascular disease mortality have declined markedly in the last 10 to 15 years, as have the risk factors of smoking, hypertension, and elevated cholesterol. Smoking reduction is particularly evident among middle-aged males, especially in those smoking <20 cigarettes/day. Smoking is an important coronary heart disease (CHD) risk factor, and individuals who quit show a substantial and fairly rapid reduction in risk of myocardial infarction and CHD death. After 10 years of followup in the Whitehall study (a smoking cessation trial), CHD mortality was 18 percent lower in the intervention group compared with controls. Also, results from the MRFIT project showed that those who quit, in both the control and the intervention groups, had a significantly decreased CHD mortality rate compared with those who continued to smoke. The risk factors of hypertension, elevated cholesterol, social class differences, diabetes, physical activity, type A behavior, medication, and the implications of the next generation of clinical trials are also discussed.

84–1460. Luoto, J. **"The Health Consequences of Smoking: Cardiovascular Disease," Summary of the 1983 Report of the Surgeon General.** *Public Health Reports* 99(1): 36-38, January-February 1984.

Evidence of the relationship between smoking and coronary heart disease (CHD) and other vascular diseases, as presented in the 1983 Surgeon General's Report, is summarized. Smokers experience a CHD mortality rate 70 percent greater than that of nonsmokers. Effects of cigarette use, which appear to be dose related, are influenced by age at initiation, length of total exposure, and depth of inhalation. Smokers also have a twofold to fourfold risk of sudden cardiac death compared with nonsmokers. In addition, cigarette smoking is associated with cerebrovascular disease, arteriosclerotic peripheral vascular disease, and among females, subarachnoid hemorrhage. Cigarette use is synergistic with elevated serum cholesterol and hypertension (the other major CHD risk factors) and displays etiological interactions with oral contraceptives. Cessation of smoking results in a greatly reduced risk over time: mortality from all causes is about 30 percent lower in ex-smokers compared with continuing smokers. It is estimated that 170,000 excess CHD deaths occur each year from cigarette smoking, yet survey data show that 53 percent of the population and 58 percent of smokers do not know that smoking is a significant factor in CHD.

84–1461. Madsen, H.; Dyerberg, J. **Cigarette Smoking and Its Effects on the Platelet—Vessel Wall Interaction.** *Scandinavian Journal of Clinical Laboratory Investigation* 44(3): 203-206, May 1984.

Results indicate that the effect of smoking on hemostasis is mediated mainly through nicotine's inhibitory effect on prostacyclin production in the vessel wall and that carbon monoxide plays only a minor role. In five habitual smokers, after smoking two high-nicotine cigarettes (2.6 mg nicotine/cigarette), mean bleeding time decreased 1.6 min ($p<0.05$), but platelet aggregability was only moderately inhibited. After inhibiting platelet cyclooxygenase by ingesting acetylsalicylic acid (ASA) 2 days prior to the study, smoking caused a markedly and significantly shortened bleeding time (-3.8 min, $p<0.05$). Bleeding time was unaffected by smoking when both platelet cyclooxygenase and vessel wall prostaglandin were inhibited by ASA 1 hour prior to the study. After smoking two cigarettes with low nicotine content (0.25 mg nicotine/cigarette) no changes in the hemostatic mechanism were observed. Inhalation of carbon monoxide in high amounts in five nonsmokers had a significant, short-lasting effect on bleeding time (-1.2 min, $p<0.05$). No changes in bleeding time were observed after carbon monoxide inhalation when platelet cyclooxygenase or both platelet cyclooxygenase and vessel wall prostaglandin production were blocked. (Auth. Abs. Mod.)

84–1462. Marmot, M. G. **Life Style and National and International Trends in Coronary Heart Disease Mortality.** *Postgraduate Medical Journal* 60(699): 3-8, January 1984.

Data from 21 countries for 1968 to 1977 indicate a small positive correlation between changes in cigarette consumption and changes in coronary heart disease (CHD) mortality. In the U.S.S.R., both CHD mortality and smoking increased between 1960 and 1978. In England and Wales, trends are stratified by income groups; both were lower in social classes I and II compared with IV and V. An international comparison of trends in CHD mortality shows a sizable decline in the United States, Australia, New Zealand, Finland, Canada, Belgium, and Japan. By contrast, in England, Wales, and Scotland, where no evidence of decline in CHD mortality was apparent until 1979, there was a marked deterioration in CHD ranking relative to other countries. Sweden, Hungary, Poland, Yugoslavia, Bulgaria, Rumania, and the U.S.S.R. have all had increases in CHD mortality. There are many possible reasons for these changes. In general, countries that had had a decrease in CHD mortality have experienced changes in the type and/or quantity of fat in the national diet, but changes in CHD mortality in Sweden and Japan are out of line with the pattern in other countries. There is some evidence that changes in smoking and blood pressure control may have contributed to changes in CHD mortality. Changes in physical activity may have also played a part. (Auth. Abs. Mod.)

84–1463. Oliver, M. F.; McKeown, T.; Lamm, G.; Morris, J. N.; Bang, H. O.; Lancet.; Kaplan, N. M.; Puska, P.; Salonen, J. T.; Tuomilehto, J.; Nissinen, A.; Koskela, K. **Coronary Risk Factors: Should We Not Forget About Mass Control?** *World Health Forum* 5(1): 5-18, 1984.

In a roundtable debate on the efficacy of mass screening and intervention programs to reduce the risk of coronary disease, it is first argued that trials to date have not provided convincing evidence of success, and while everyone should be urged to quit smoking, the strategy for preventing disease needs to be reconsidered, concentrating on those persons identified as being at particularly high risk. Critics of such an approach cite inconsistencies in the way data were interpreted and problems inherent in restricting intervention to high risk groups only, particularly because of difficulties in identifying those at such risk. It is suggested that the prevention approach could be altered to "guidelines for healthy living," which would include changes in diet and exercise, in addition to cessation of smoking, for the total population. The significance of dietary changes (*Lancet* editorial reprinted) on a national level is discussed, noting that mass screening and high-risk screening are not necessarily mutually exclusive. Several comments in defense of mass screening are given, suggesting that public health decisions on risk factor reduction must be based on broad information on the overall effects of such efforts, which a community study can help provide. The original author defends his premise in comments about the other stated positions.

84–1464. Rao, R. S. **Influence of Smoking on Blood Pressure.** *Indian Journal of Medical Sciences* 37(7): 115-119, July 1983.

Data on the influence of smoking on blood pressure (BP) among rural, semiurban, and urban populations of a coastal Indian province showed that, as a general trend, nonsmokers ($n=716$) had higher mean systolic and diastolic BP compared with smokers ($n=866$). Subjects were classified as current smokers, never smokers, and ex-smokers who had been abstinent for at least 1 year. Chi-square analyses revealed significant associations between smoking status and diastolic BP, but no association between smoking status and systolic BP. Although the findings contribute to a better understanding of the impact of smoking on BP, further research on hypertensive patients is urgently needed among Indian populations.

84–1465. Sivarajan, E. S.; Newton, K. M. **Exercise, Education, and Counseling for Patients With Coronary Artery Disease.** *Clinics in Sports Medicine* 3(2): 349-369, April 1984.

The role of smoking cessation in a multifactorial cardiac rehabilitation program that incorporates education, exercise, and counseling is discussed. It is noted that smoking is the risk factor for which the benefits of secondary prevention are most conclusive. In one study, 5-year mortality was 16 percent among ex-smokers compared with 63.6 percent among continuing smokers. The infarction itself often provides the motivation necessary to quit without special intervention. However, specific smoking intervention begun during hospitalization results in twice as many patients giving up smoking as controls; patients who decide to quit while hospitalized appear to abide by the decision. When quit-smoking advice is part of an organized program of cardiac rehabilitation, abstinence rates of 78.1 percent have been achieved. Besides smoking cessation, issues surrounding supervised vs. unsupervised exercise, dietary changes, stress management, high blood pressure, and returning to work are reviewed. Discussion of the specific needs of female cardiac patients and a prototype rehabilitation program are also included.

84–1466. Squires, W. G., Jr.; Brandon, T. A.; Zinkgraf, S.; Bonds, D.; Hartung, G. H.; Murray, T.; Jackson, A. S.; Miller, R. R. **Hemodynamic Effects of Oral Smokeless Tobacco in Dogs and Young Adults.** *Preventive Medicine* 13(2): 195-206, March 1984.

Results indicate that oral smokeless tobacco use can produce significant hemodynamic changes in both dogs and normal humans. To determine the cardiovascular effects of such a product, 10 anesthetized dogs were instrumented to measure blood pressure, heart rate, left ventricular end diastolic pressure, and circumflex coronary, renal, and femoral flows. After a 5-min baseline, a 2.5 g (approximately 1.2 percent nicotine) bolus dose was placed in the buccal space and measurements were made for 20 min. Significant increases were seen in heart rate, blood pressure, left ventricular pressure, left ventricular end diastolic pressure, and left ventricular dP/dt. Significant decreases in flow were noted in the coronary circumflex, renal, and femoral arteries. Flow reduction was thought to have been mediated by an α-adrenergic mechanism. Additionally, 20 human males, mean age 20 years, without nicotine exposure for 72 hr, were given a 2.5 g dose of the same oral smokeless product. From baseline to 20 min, heart rate increased from 69 to 88 beats/min ($p<0.05$), and blood pressure from 118/72 to 126/78 mm Hg ($p<0.05$). (Auth. Abs. Mod.)

84–1467. Stemmermann, G. N.; Hayashi, T.; Resch, J. A.; Chung, C. S.; Reed, D. M.; Rhoads, G. G. **Risk Factors Related to Ischemic and Hemorrhagic Cerebrovascular Disease at Autopsy: The Honolulu Heart Study.** *Stroke* 15(1): 23-28, January-February 1984.

Hypertension, cigarette use, and alcohol consumption were strongly associated with intracerebral hemorrhagic disease in Hawaiian Japanese men. Cerebral infarcts accompanied myocardial infarction (MI) in 58 percent of cases and were associated with high serum cholesterol, hypertension, and severe atherosclerosis of the coronary arteries and aorta. Smoking incidence, however, did not vary significantly between the living, the brain infarct with MI subjects, and the brain infarct without MI subjects, although an increasing trend was observed. However, significant associations did not persist when coronary heart disease cases were removed from the analysis, indicating the presence of two subsets of men with cerebral infarction. (Auth. Abs. Mod.)

84–1468. Wagrowska, H.; Balaz, V.; Rywik, S.; Korczynska, M.; Bederova, A. **Prevalence of Ischaemic Heart Disease Risk Factors in Male Population Aged 45-54 Years in Warsaw and Bratislava. Part III: Smoking and Overweight.** *Cor et Vasa* 25(5): 329-338, 1983.

Prevalence of ischemic heart disease (IHD) risk factors such as cigarette smoking and obesity and the degree of IHD risk were investigated in Polish and Slovak populations. Subjects were men aged 45 to 54, 2,944 from Warsaw industrial plants and 698 from Bratislava plants. Among Warsaw subjects, 60.4 percent were current smokers compared with 46.4 of Bratislava subjects. The proportion of smokers decreased with increasing age in both populations, but there were significantly fewer ex-smokers than in other European countries, suggesting the relative ineffectiveness of Polish and Czech antismoking campaigns. Subjects in Bratislava were significantly more overweight, and the IHD risk, evaluated according to the multilogistic function, was higher than in Warsaw workers. It is concluded that the difference in IHD death rate found in these cities could be explained largely by the difference in the prevalence of risk factors. (Auth. Abs. Mod.)

84–1469. Wood, C. **Heart Disease—Action Now, Proof Later.** *New Scientist* 98(1355): 214-217, April 28, 1983.

Data show that regardless of blood cholesterol or systolic blood pressure levels, smokers are at much higher risk of first heart attack than nonsmokers. The key question in the prevention of heart attacks in middle-aged men prone to coronary heart disease (CHD) is whether, after decades of exposure to various risk factors such as smoking, it is possible to reduce the frequency of such events (men have about four times as many heart attacks as women). Major CHD studies have not provided convincing evidence that intensive intervention actually reduces the frequency of disease. Typically, results of the North Karelia Project showed that reduction in risk factors occurred in both intervention and control groups, probably due in part to changes in attitudes in the general population, such as those toward smoking. Control groups could not be stopped from doing what they saw others doing. The Stanford Five Counties Study, described as an ambitious example of community intervention, will run through 1986 at a cost of about 10 million dollars. The goal is to reduce coronary risk by 20 percent by getting smoking, blood pressure, cholesterol, and body weight down by amounts ranging from 2 to 9 percent. The multimedia approach includes television and radio spots; newspapers and magazine articles; books and pamphlets on diet, blood pressure, and exercise; how to quit smoking kits; T-shirts; and lapel buttons. The World Health Organization and other international groups recommend such actions worldwide, but whether it will happen will depend on the zeal and commitment of the believers, how resistant the population is to change, and how much hostility is shown by those with vested interests (such as the tobacco industry, which would face slow extinction).

84–1470. Yano, K.; Reed, D. M.; McGee, D. L. **Ten-Year Incidence of Coronary Heart Disease in the Honolulu Heart Program. Relationship to Biologic and Lifestyle Characteristics.** *American Journal of Epidemiology* 119(5): 653-666, May 1984.

Of 14 biologic and lifestyle factors measured at baseline examination, systolic blood pressure was the most potent and consistent risk factor for all categories of coronary heart disease (CHD) except uncomplicated angina. Cigarette smoking was closest to systolic blood pressure as a potent and consistent risk factor for total CHD, fatal CHD, and acute coronary insufficiency, and showed an even stronger association with nonfatal myocardial infarction. Data are from 10 years of followup of 7,705 Japanese men living in Hawaii (45 to 68 years of age and free of disease at initial examination). A total of 511 new CHD cases were identified. The incidence rate of fatal CHD and nonfatal myocardial infarction was less than half the rate for whites in the United States and about twice the rate for Japanese men in Japan. Other factors significantly associated with CHD were serum cholesterol and age. Alcohol consumption demonstrated a strong protective effect on fatal CHD and nonfatal myocardial infarction.

See also **84–1336**

PREGNANCY AND INFANT HEALTH

84–1471. Busacca, M.; Balconi, G.; Pietra, A.; Vergara-Dauden, M.; de Gaetano, G.; Dejana, E. **Maternal Smoking and Prostacyclin Production by Cultured Endothelial Cells From Umbilical Arteries.** *American Journal of Obstetrics and Gynecology* 148(8): 1127-1130, April 15, 1984.

Endothelial cells from umbilical cords of mothers who smoked produced less prostacyclin (PGI_2) than cells from nonsmokers, although the only significant difference was between heavy smokers and never smokers. Cells were obtained from 22 apparently healthy women, 11 mild smokers (<15 cigarettes/day), and 25 heavy smokers (>15 cigarettes/day). Production of PGI_2 by the cells was calculated by measuring the amount of 6-keto-prostaglandin $F_{1\alpha}$ released in the culture medium after addition of arachidonic acid, by means of a specific radioimmunoassay. In addition to lowered PGI_2 production, cell cultures from smokers showed a lower success rate: 23 percent of cultures from heavy smokers and 64 percent from mild smokers were successful, compared with a 77 percent success rate in cultures from nonsmokers. Smoking during pregnancy appears to induce some modifications in the enzymes of the prostacyclin pathway that persist in endothelial cells undergoing replication in primary culture even in the absence of the pathogenic factor. (Auth. Abs. Mod.)

84–1472. Cederqvist, L. L.; Eddey, G.; Abdel-Latif, N.; Litwin, S. D. **The Effect of Smoking During Pregnancy on Cord Blood and Maternal Serum Immunoglobulin Levels.** *American Journal of Obstetrics and Gynecology* 148(8): 1123-1126, April 15, 1984.

The effect of smoking on the fetal and maternal humoral immune parameters was evaluated in cord and maternal blood, collected at delivery from 163 mothers who smoked and 130 mothers who did not smoke and from their offspring. There was no difference in time of gestation between the two groups, but mean birth weight was decreased in offspring of mothers who smoked, as previously observed. There were higher levels of immunoglobulin A (IgA, $p<0.01$), immunoglobulin M (IgM, $p<0.001$), and immunoglobulin G (IgG, $p<0.001$) in cord sera of children of mothers who smoked than in offspring of nonsmokers. Mothers who smoked had higher levels of IgM ($p<0.001$) and IgG ($p<0.001$), but not IgA, compared with nonsmoking control mothers. Since cord IgA and IgM are produced by the fetus, results could be interpreted as being due to either a higher incidence of infection in utero or metabolic differences in children of mothers who smoke. Either of these possibilities may explain the increased frequency of postpartum endometritis, increased incidence of fetal distress, and the characteristic of meconium-stained amniotic fluid in mothers who smoke. (Auth. Abs.)

84–1473. Cito, G.; Buonocore, G.; Monti, E.; Severi, F. M.; Vanni, A. L. **Correlazione fra Livelli di Carbossiemoglobina Materna, Frequenza Cardiaca e Movimenti Fetali Prima e Dopo il Fumo di Sigaretta (Smoking Test). [Correlation Between Maternal Carboxyhemoglobulin Levels, Heart Rate, and Fetal Movement Before and After Cigarette Smoking.]** *Clinica* 38(5/6): 245-251, 1983, Italian.

Continuous cardiographic monitoring and carboxyhemoglobin (COHb) analyses were performed on 10 pregnant women with normal pregnancies (between 33 and 38 weeks). Patients abstained from smoking for 8 hours and then smoked a cigarette. Blood samples were obtained before, during, and after smoking. A progressive increase in the percentage of COHb was observed for the initial 30 minutes after beginning smoking. Cardiographic monitoring showed an early acceleration of the fetal heart rate, followed by a lowering of the heart rate and fetal movements. In only one case (a pregnancy of 38 weeks) were uterine contractions observed after smoking. These were associated with a late deceleration of the fetal heart rate from 175 to 140 beats/minute. It is recommended that obstetric patients avoid smoking in the third trimester due to the high COHb blood levels that persist for hours after smoking, which increase the risk of fetal hypoxia and uterine contractions. (Auth. Abs. Mod.)

84–1474. Garn, S. M.; Ridella, S. A.; Smith, B. H.; Hingson, R. **Problems of Sample Size and Selection in Analyses of Pregnancy Outcomes. (Letters).** *American Journal of Obstetrics and Gynecology* 147(7): 852-854, December 1, 1983.

Low Apgar scores reported in a previous study (*American Journal of Obstetrics and Gynecology* 144(8): 959-966, December 15, 1982) are questioned, suggesting that small sample size, ethnic origin, and age could have masked a smoking effect. While low Apgar scores do not reflect maternal smoking as dramatically as low birth weight, the far larger Collaborative Perinatal Project found that low scores are more frequent in babies of mothers who smoke. In reply, a fundamental difference in data analysis in the two studies (univariate and bivariate vs. multiple regression procedure) is discussed. It is suggested that other maternal characteristics (illness before or during pregnancy, alcohol consumption, use of psychoactive substances) or other independent effects of labor and delivery might have contributed to the differences in reported findings. While low Apgar scores were not found to be indicative of maternal smoking, the proven relationship with low birth weight still makes smoking a habit to be discouraged during pregnancy. See also 84-0556.

84–1475. Gusella, J. L.; Fried, P. A. **Effects of Maternal Social Drinking and Smoking on Offspring at 13 Months.** *Neurobe-*

havioral Toxicology and Teratology 6(1): 13-17, January-February 1984.

Data on the relationship between prenatal alcohol/nicotine consumption and developmental infant performance showed that maternal smoking was related to a decrease in motor skills and verbal comprehension. Using the Bayley Mental and Motor Scales as indices of infant development, 84 infants aged 13 months were evaluated. Smoking prior to pregnancy was related to a lower average Psychomotor Development Index (PDI, $r = -0.37$, $p < 0.001$), fine motor skills ($r = -0.27$, $p < 0.01$), Motor Developmental Index ($r = -0.20$, $p < 0.05$), and verbal comprehension ($r = -0.27$, $p < 0.01$). Smoking during pregnancy was related to a decrease in PDI ($r = -0.19$, $p < 0.05$), and verbal comprehension ($r = -0.31$, $p < 0.01$). After adjusting for father's educational level, pre-pregnancy smoking was related only to fine motor skills and smoking during pregnancy to verbal comprehension. Maternal alcohol consumption, at "social" levels, was related to a decrease in motor scale scores, verbal comprehension, and fine motor skills, after adjusting for confounding variables. No significant relationship with infant temperament was found, and there was no significant correlation between maternal alcohol and nicotine consumption.

84–1476. Kaelin, A. C.; Cummings, A. J. **A Survey of Aryl Hydrocarbon Hydroxylase Activity in Human Placental Homogenates.** *Placenta* 4(Special issue): 471-478, 1983.

Average placental levels of aryl hydrocarbon hydroxylase (AHH) were 50 to 60 times greater in smokers ($n = 61$) compared with nonsmokers ($n = 44$) in a study of monooxygenase activity in vitro toward 7-ethoxyresorufin (ERR) and 2,5-diphenyloxazole (PPO). Some association was found between levels of placental AHH activity and the number of cigarettes smoked/day ($p < 0.01$). In smokers, placental PPO hydroxylase and ERR O-deethylase activities were significantly correlated ($r = 0.96$, $p < 0.001$). In nonsmokers, the low average levels of AHH activity obtained using both substrates were found to be significantly non-zero: PPO, $p < 0.01$; ERR, $p < 0.001$. No correlation was found, in either smokers or nonsmokers, between placental AHH activity, age, body weight, diet (including consumption of tea, coffee, and alcohol), number of previous pregnancies, or placental and baby birth weights. Smokers who reduced consumption during pregnancy still maintained fairly high levels of AHH activity. Among epileptics receiving anticonvulsive therapy ($n = 3$), administration of carbamazepine appeared to cause some enhancement of placental AHH activity, but phenytoin had no apparent effect. (Auth. Abs. Mod.)

84–1477. Kelly, J.; Mathews, K. A.; O'Conor, M. **Smoking in Pregnancy: Effects on Mother and Fetus.** *British Journal of Obstetrics and Gynaecology* 91(2): 111-117, February 1984.

Significant differences in maternal and fetal effects of smoking tobacco vs. nontobacco cigarettes were found in 75 pregnant patients compared with matched controls (22 pregnant nonsmokers). Patients ($n = 50$) who smoked a New Smoking Materials Ltd. (NSM) cigarette followed by a tobacco cigarette (Protocol 1) showed no significant differences in maternal heart rate, maternal mean arterial pressure, fetal heart rate, and fetal heart rate "baseline variability" before and after smoking the cigarette. However, there was a significant increase in maternal heart rate, maternal mean arterial pressure, and fetal heart rate, and a significant decrease in fetal heart rate baseline variability, after smoking the tobacco cigarette. Patients ($n = 25$) who smoked two NSM cigarettes sequentially (Protocol 2) showed no significant change in maternal heart rate, maternal mean arterial pressure, fetal heart rate, or fetal heart rate baseline variability. However, when the same patients smoked two tobacco cigarettes sequentially, there was a significant increase in maternal heart rate, maternal mean arterial blood pressure, and fetal heart rate, accompanied by significant decreases in fetal heart rate baseline variability. There were also significant differences when NSM smokers and tobacco smokers were compared with nonsmokers. Since the observed changes were more marked with tobacco smoking than with NSM smoking, results suggest the main effects of cigarettes are due to the nicotine content. (Auth. Abs. Mod.)

84–1478. Murphy, J. F. **Drugs and Pregnancy.** *Irish Medical Journal* 77(2): 52-56, February 1984.

Cigarette smoking during pregnancy adversely affects the developing fetus, with reported hazards such as increased incidence of spontaneous abortion, intrauterine growth retardation and prematurity, and increased perinatal mortality. On average, babies of smoking mothers weigh 150 to 400 g less than babies of nonsmokers, and twice as many weigh less than 2,500 g. While it is still not clear whether such growth retardation extends into postnatal life, some differences in height and reading attainment at age 7 were reported in one study of over 5,000 British children. Extended research using the same sample indicated similar problems at age 11. The exact mechanism of action for these effects of maternal smoking remains to be established in most cases. Meanwhile, international efforts to control smoking should continue, and women should be advised to quit smoking, particularly during pregnancy.

84–1479. Nilsen, S. T.; Sagen, N.; Kim, H. C.; Bergsjo, P. **Smoking, Hemoglobin Levels, and Birth Weights in Normal Pregnancies.** *American Journal of Obstetrics and Gynecology* 148(6): 752-758, March 15, 1984.

A strong correlation was found between smoking and low birth weight among 811 Norwegian women studied during the last trimester of normal singleton pregnancies. Among women who smoked during the third trimester, no significant association existed between smoking habits and hemoglobin levels, but the relatively few heavy smokers (> 10 cigarettes/day) may have precluded discovery of subtle differences. Birth weights were transferred to a weight-for-dates percentile scale, based on distribution of the total Norwegian birth population over an 11-year period. When grouped by quartiles according to this scale, 47 percent of newborn infants of heavy smokers were in the lowest quartile. Hemoglobin levels were inversely correlated with birth weight quartiles in both smoking and nonsmoking mothers. However, when birth weights were grouped by maternal hemoglobin at term, the tobacco-related effect on birth weight appeared only with hemoglobin levels above 12 gm/dl, particularly for levels above 13 gm/dl. Hemoglobin levels of 9.0 to 11.0 gm/dl appeared to prevent growth retardation in fetuses of nonanemic mothers who smoked. Ponderal, or weight-by-length, indices showed growth-retarded babies of mothers who smoked tended to be thin, indicating possible nutrient deprivation. (Auth. Abs. Mod.)

84–1480. Pelkonen, O.; Pasanen, M. **Enzymology and Regulation of Xenobiotic and Steroid Metabolism in Placenta.** *Biochemical Society Transactions* 12(1): 42-44, February 1984.

Current concepts on the enzymology of placenta xenobiotic and steroid metabolism and the unique aspects of the response to exogenous influences such as tobacco smoke are reviewed. Among classical inducers, only cigarette smoke induces placental xenobiotic metabolism. Compared with other human tissues, the placenta is clearly extraordinary in its response to maternal cigarette smoking. Although tissue and culture differences have popularized the placenta for molecular-biological studies, a major difficulty has been the inability to characterize the benzo(*a*)pyrene-metabolizing cytochrome P-450. Data also suggest that the environment affects placental steroid metabolism. An inverse correlation between aryl hydrocarbon hydroxylase and cholesterol side-chain cleavage has been demonstrated in placentas from smokers, while aromatase activity seems affected and catechol estrogen formation is stimulated. Findings suggest that although steroid metabolism is steadily regulated, exogenous pharmacological or toxicological influences may have some effect, the mechanism of which remains to be elucidated.

84–1481. Prager, K.; Malin, H.; Spiegler, D.; Van Natta, P.; Placek, P. J. **Smoking and Drinking Behavior Before and During Pregnancy of Married Mothers of Live-Born Infants and Stillborn Infants.** *Public Health Reports* 99(2): 117-127, March-April 1984.

Unweighted data from the 1980 National Natality Survey and the National Fetal Mortality Survey (4,405 mothers of live-born and 2,616 mothers of stillborn infants, all married) show that 34 percent of mothers of stillborns smoked before pregnancy compared with 31 percent of mothers of live-borns. Among mothers over age 33, figures were 31 and 21 percent, respectively. Only 16 percent of mothers of stillborns stopped smoking during pregnancy, compared with 30 percent of mothers of live-born infants. White mothers under age 25 with a high school education or less were more apt to be smokers than were black or Hispanic mothers. White mothers 25 years of age and older with more than a high school education were more apt to be drinkers. Reductions in smoking and drinking occurred when pregnancy was confirmed. Although the prevalence of drinking was much higher than smoking, the reduction in drinking was much more pronounced than the reduction in smoking. Differences in alcohol consumption between mothers of live-born and mothers of stillborn infants were small. About 22 percent of white mothers both smoked and drank, compared with about 13 percent of black mothers and 14 percent of Hispanic mothers. The combination of behaviors decreased with increased age and education for all ethnic groups. Controlled multivariate analysis will be necessary to specify the relationship of maternal smoking and drinking with birth outcome.

84–1482. Stetson, D. E.; Andrasik, F. **Acute Effects of Cigarette Smoking on Pregnant Women and Nonpregnant Control Subjects.** *American Journal of Obstetrics and Gynecology* 148(6): 794-798, March 15, 1984.

The effect of cigarette smoking on pulse rate and carboxyhemoglobin concentration was studied in seven healthy pregnant smokers (≥ 10 cigarettes/day for ≥ 6 years) during weeks 11-26 of gestation to (1) standardize cigarette consumption for studying short-term smoking effects in pregnant smokers; (2) test for these effects by use of two noninvasive measures (concentration of carboxyhemoglobin in expired air and digital pulse rate); and (3) compare the acute effects evidenced by pregnant smokers with those of a carefully matched group of nonpregnant control subjects. Increases in pulse rate and concentration of carboxyhemoglobin were positively correlated with nicotine intake and concentration of inhaled mainstream smoke, respectively. Results show no evidence of significant differences between pregnant and nonpregnant women on either of the smoking measures. Methodologic implications of these findings are discussed.

84–1483. Ventura, S. J.; Hendershot, G. E. **Infant Health Consequences of Childbearing by Teenagers and Older Mothers.** *Public Health Reports* 99(2): 138-146, March-April 1984.

Data from the National Natality Survey and the National Fetal Mortality Survey (1980) showed that nearly 90 percent of women over the age of 30 who experienced a first birth were employed, 48.7 percent in professional categories. The smoking habits of the women differed significantly according to occupation; professional women were least likely to smoke before or during pregnancy. However, statistics on nonsmokers show an association between smoking and pregnancy outcome regardless of employment status or occupational category. Although analysis is limited by the small number of births involved, it appears that professionally employed women in the over 30 age group generally have the best pregnancy outcomes, especially nonsmokers. Survey data on the effect of marital status on pregnancy outcome among teenaged mothers is also provided.

84–1484. Williams, C. M.; Kanagasabai, T. **Maternal Adipose Tissue Response to Nicotine Administration in the Pregnant Rat: Effects on Fetal Body Fat and Cellularity.** *British Journal of Nutrition* 51(1): 7-13, January 1984.

Since nicotine has been implicated as a causative factor in intrauterine growth retardation associated with smoking during pregnancy, the effect of nicotine on fetal growth and possible relationships between nicotine's actions and maternal adipose tissue metabolism were studied in rats. Sprague-Dawley rats were mated and assigned to control and nicotine groups, the latter receiving nicotine in drinking water throughout pregnancy. Animals were weighed at regular intervals and killed on day 20. Rates of maternal adipose tissue lipolysis and lipogenesis were measured, fetal and placental weights were recorded, and fetal body water, fat, protein, and DNA were analyzed. Maternal weight gains in the nicotine group were less in weeks 1 and 2 of pregnancy, but similar to controls in week 3. Fetal body weights, DNA, protein, and percentage water contents were similar in both groups. Mean fetal body fat (g/kg) was significantly higher in the nicotine group compared with controls, and maternal lipolysis rates were also higher. The cause of these differences and their effects on maternal and fetal well-being are discussed. It is concluded that intrauterine growth retardation associated with maternal smoking may not be due to nicotine, although its actions during pregnancy may have important nutritional consequences to offspring and mother. (Auth. Abs. Mod.)

See also, **84–1329, 84–1331, 84–1350**

OTHER DISEASES AND CONDITIONS

84–1485. Bosse, R.; Rose, C. L. **Smoking and Aging.** Lexington, Massachusetts, D.C. Heath and Company, Lexington Books, 1984, 269 pp.

This critical analysis of the relationship between smoking and aging, with details of age/smoking interactions on various functions, disorders, and diseases, includes information on methodological considerations of aging research and the impact of aging on behavioral aspects of smoking, impact of smoking on drug metabolism, and efficacy of smoking cessation strategies. Written for investigators in the field, health professionals, and informed laypersons, it addresses issues in terms of statistical associations rather than basic mechanisms, covering both behavioral and biomedical aspects. Specific topics discussed are (1) conceptual and methodological aspects of smoking and aging, an introduction that provides a general background for critical appraisal of the literature; (2) smoking and aging effects on oral health, which describes specific age-related, nonpathological physiological phenomena, and acute and chronic diseases, and how they are affected by smoking; (3) the progressive decline in pulmonary function with age and the effect of environmental risk factors, particularly cigarette smoking, that can modify normal lung function; (4) chronic bronchitis, asthma, and obstructive airways disease; (5) coronary heart disease, the leading cause of death in the United States, and the increased mortality risk with age and daily smoking consumption level; (6) epidemiological aspects of aging, mortality, and smoking; (7) smoking, aging, and drug metabolism; (8) psychosocial aspects of smoking behavior throughout the life cycle; (9) age, health status, and other factors in smoking cessation; (10) age variance in the efficacy of smoking cessation strategies; and (11) an overview of the field, focusing on smoking and aging rather than on smoking and disease, and the further research needed on smoking, health, and aging. The source for much of the information was the Normative Aging Study and the Dental Longitudinal Study, begun in 1963 by the Veterans Administration.

84–1486. Christensen, S. B.; Ericsson, U.-B.; Janzon, L.; Tibblin, S.; Melander, A. **Influence of Cigarette Smoking on Goiter Formation, Thyroglobulin, and Thyroid Hormone Levels in Women.** *Clinical Endocrinology and Metabolism* 58(4): 615-618, April 1984.

The influence of cigarette smoking on goiter formation, thyroglobulin (Tg) secretion, and thyroid hormone production was assessed in 441 randomly selected women aged 48 to 53. Radioimmune assay of Tg, T_3, rT_3, T_4, and thyroid-stimulating hormone (TSH) were done on sera of 192 never smokers, 80 ex-smokers, and 169 smokers (121 moderate smokers, 1-19 cigarettes/day; 48 heavy smokers, ≥ 20 cigarettes/day). Palpable goiter was found in 15 percent of the smokers, 4 percent of ex-smokers, and 9 percent of never smokers. Tg values >30 µg/l were found in 37, 16, and 18 percent, respectively. Smokers had higher serum T_3 and lower rT_3 concentrations than never smokers, with the greatest difference in heavy smokers. T_4 levels did not differ, and TSH was insignificantly lower in smokers compared with nonsmokers. Levels in ex-smokers resembled those of nonsmokers. It is concluded that smoking may be goitrogenic (possibly owing to inhaled thiocyanate) and may also enhance the formation of T_3 at the expense of rT_3 formation. (Auth. Abs. Mod.)

84–1487. Grant, E.C.G. **Migraine, Headache, and Survival in Women. (Letter).** *British Medical Journal* 287(6406): 1718, December 3, 1983.

The unexpected higher mortality in women aged 45 to 64 who did not have headaches or migraine, compared with those who suffered from such ailments, may occur because women who are susceptible to migraine are less likely to have ever smoked. A survey of 336 migraine patients at a British clinic indicated significant differences between smokers and nonsmokers. Smokers had more frequent and severe attacks, were more likely to require hospitalization for acute attacks, and exhibited a 10-fold decrease in the number of attacks following cessation of smoking. In this sample, 74 percent of the patients were women and eight times more women than men over age 40 had never smoked, suggesting an early intolerance.

84–1488. Houston, T. P. **Women Who Smoke: An Equal Right to Die.** *American Family Physician* 29(3): 128, 131, March 1984.

Smoking-related mortality and morbidity among women in the United States is discussed, noting that lung cancer mortality has increased from 4.6/100,000 in 1950 to an estimated 20.9/100,000 in 1982, and will probably surpass breast cancer as the leading cause of cancer mortality in women by the end of 1984. The risk of myocardial infarction in those smoking one pack or more a day is 10 times that found in women who never smoked. Advertising targeted at women, particularly of low-nicotine cigarettes, and lack of articles on the health hazards of smoking in popular women's magazines may help explain the increased incidence of smoking. It is suggested that the tobacco industry is powerful enough to discourage periodicals that accept tobacco advertising from publishing health-related information. The large number of women smoking low-nicotine cigarettes (a higher proportion than among men) may feel they are less hazardous, although published tar and nicotine levels have been shown to be invalid, since many smokers compensate by changing smoking behavior.

84–1489. Kawane, H. **Drinking and Passive Smoking. (Letter).** *Chest* 85(3): 452, March 1984.

Comments on a recent special report on the health risks of passive smoking focus on the serious disregard for the rights of nonsmokers in Japanese public places. Japanese are permitted to smoke on trains, in recreational facilities, and at work. No provisions are made for special smoking areas. Smoking in bars is a particular problem, and the functional pulmonary impairment observed in a study of alcoholics (after correcting for cigarette smoking) may reflect the effects of passive smoking. Moderate-to-heavy drinkers tend to stay longer in bars, pubs, or saloons, where there are high smoke levels.

84–1490. Ketkar, M. B.; Fuhst, R.; Mohr, U. **The Effect of Cigarette Smoke Exposure in European Hamsters.** *Experimental Pathology* 25(2): 103-106, 1984.

European hamsters ($n=60$) were exposed in a chamber to the smoke of one experimental cigarette/day, 5 days/week, for 130 weeks. Two types of cigarettes were used: C1, of Virginia tobacco and with high tar and nicotine yields; and C2, cigarettes made of reconstituted tobacco and having lower yields. Cigarettes were smoked at one puff/minute, 35 ml/puff, with a puff duration of 2 seconds. Exposure to either type of cigarette smoke produced a 25 percent reduction in body weight compared with 29 untreated controls. Also, exposure-related inflammatory reactions, hyperplasias, and metaplasias of the forestomach and respiratory tract were found. However, no neoplastic responses or effects on mortality were seen.

84–1491. Larsson, L.; Orlander, J. **Skeletal Muscle Morphology, Metabolism and Function in Smokers and Non-Smokers. A Study on Smoking-Discordant Monozygous Twins.** *Acta Physiologica Scandinavica* 120(3): 343-352, March 1984.

Smoking produces changes in muscle fiber type distribution and associated muscle characteristics. Six pairs of smoking-discordant monozygous twins were studied with respect to muscle (vastus lateralis) morphology, metabolism, and function. The percentage of type I fibers was lower in smokers, who also had smaller diameters of this fiber type. Cytochrome oxidase activity was decreased in smokers. No differences were seen for other enzymes of energy metabolism, capillary density, isometric or dynamic strength, or short-term muscular endurance. Nonsmokers tended to be more physically active. In four ex-smoker/nonsmoker pairs, no significant differences were found for the investigated parameters. It was concluded that the difference in fiber type distribution is not due to a hereditary predisposition to take up smoking or to the small difference in physical activity level. Thus, smoking per se appears to be the most plausible explanation for the difference in fiber type distribution and associated muscle characteristics. No definitive conclusion regarding the reversibility of the smoking-related differences can be drawn from present results. (Auth. Abs. Mod.)

84–1492. Lawrence, W. T.; Murphy, R. C.; Robson, M. C.; Heggers, J. P. **The Detrimental Effect of Cigarette Smoking on Flap Survival: An Experimental Study in the Rat.** *British Journal of Plastic Surgery* 37(2): 216-219, April 1984.

Flap survival was significantly decreased in 10 rats exposed to cigarette smoke compared with 8 nonexposed rats. A smoke chamber was constructed to evaluate the effect of intermittent cigarette smoke exposure on the survival of a McFarlane flap on the dorsum of a rat. Oxygen and carbon dioxide levels and temperature, monitored in the chamber during smoke exposures, consistently approximated values in room air. Average flap survival at 7 days in control rats not exposed to cigarette smoke was 75.2 percent, while flap survival decreased to 40.1 percent in rats intermittently exposed to cigarette smoke. The difference was statistically significant ($p<0.01$). Changes in carboxyhemoglobin levels similar to those observed in human smokers were also found in smoke-exposed rats. Findings indicate that postoperative cigarette smoking is detrimental to flap survival. (Auth. Abs. Mod.)

84–1493. Logan, R.F.A.; Edmond, M.; Somerville, K. W.; Langman, M.J.S. **Smoking and Ulcerative Colitis.** *British Medical Journal* 288(6419): 751-753, March 10, 1984.

Results of a case—control study of smoking and ulcerative colitis showed that patients with the disease were much less likely to smoke than community controls matched for age and sex. The difference was substantial, with an estimated relative risk of 3.8 for current nonsmoking status. It was even larger (6.2) when smoking habits at onset of the disease were examined, and was mainly accounted for by 42 of 55 patients who had given up smoking a mean of 8 years before onset. The association could not be explained by confounding by social class. Findings suggest that smoking directly or indirectly confers protection against ulcerative colitis. (Auth. Abs.)

84–1494. Medico-Social Research Board. **The Health Effects of Smoking in Ireland.** In: *Annual Report 1982.* Dublin, Ireland, The Medico-Social Research Board, 1983, pp. 74-78.

Data on the health effects of smoking in Ireland indicate that coronary heart disease (CHD) is the chief cause of excess mortality among cigarette smokers, particularly in middle age. Total deaths caused by CHD increased from 7,262 in 1968 to 8,846 in 1977. Deaths caused by cancer of the trachea, bronchus, and lung have increased steadily for men and women in recent decades, reflecting the increase in cigarette smoking, particularly among men, in the mid-1940s and early 1950s. In addition to premature deaths, smoking is associated with chronic diseases such as stroke, blood vessel disease, and chronic bronchitis/emphysema, creating a burden for the community and causing many hospital admissions. Lower birth weight is common in infants of smoking mothers, and the effects of passive smoking in the home or work environment are also becoming evident. It is hoped that these observations will induce Irish policy makers and health educators to adopt more stringent antismoking programs.

84–1495. Miller, G. H. **Passive Smoking—Corrections in Tables of April Article. (Letter).** *Western Journal of Medicine* 140(6): 956, June 1984.

A table on causes of death in all women in relation to husband's smoking history, which contained several typographical errors when first printed (*Western Journal of Medicine* 140(4): 632-635, April 1984) is reproduced. Corrected percentages for other errors in tabular data from the earlier report are also provided. See also 84-1392.

84–1496. Patten, B. M. **Neuromuscular Disease Due to Tobacco Use.** *Texas Medicine* 80: 47-51, April 1984.

Neuromuscular symptoms in five tobacco users are described, and reports of nicotine toxicity in the literature are reviewed. Although several factors, including individual susceptibilities, affect a person's nicotine level, subclinical neuromuscular toxicity is probably not rare among cigarette smokers and users of chewing tobacco and snuff. Nicotine poisoning can cause neuropathy in patients with chronic obstructive pulmonary disease. It may also be implicated in myasthenic syndrome associated with desensitization of postsynaptic membrane to the normally depolarizing action of the neurotransmitter acetylcholine. In addition to toxic effects on the neuromuscular apparatus, nicotine has adverse effects on the heart, blood vessels, and circulation. In view of current evidence, physicians should consider nicotine toxicity in all tobacco users who have unexplained weakness or fatigability.

84–1497. Rajput, A. H. **Epidemiology of Parkinson's Disease.** *Canadian Journal of Neurological Sciences* 11(1, Supplement): 156-159, February 1984.

Although a negative association between smoking and parkinsonism has been suggested in previous studies, with some even suggesting that smoking has a protective effect against the disease, data for 138 cases in Rochester, Minnesota, showed smoking habits prior to onset of parkinsonism were similar for patients and controls. It is therefore concluded that smoking does not have a protective effect. The annual incidence rate was 20.5/100,000, virtually unchanged from previous reports. Prior to diagnosis, dementia was diagnosed significantly more frequently among patients than controls. After diagnosis, dementia emerged nearly three times more frequently in patients than controls. Mortality rate in the parkinsonian population was similar to that reported between 1935 and 1966. Among those treated with levodopa, the mortality rate was comparable with controls. Because there were no definite criteria for administering levodopa therapy, higher frequency of dementia and prolonged survival in those treated with this drug could be attributed to the bias of case selection for treatment. (Auth. Abs. Mod.)

84–1498. Repace, J. L. **Tobacco Smoke and the Nonsmoker.** Presented at the Fifth World Conference on Smoking and Health, Winnipeg, July 10-15, 1983, 17 pp.

Studies on the quantification of nonsmoker exposure to ambient tobacco smoke are reviewed, and a single-compartment model for the translation of exposure into dose is proposed. The model yields an estimated range of equilibrium lung doses of tobacco tar from 5 mg to 160 mg for nonsmokers who are exposed on a daily basis. The average nonsmoker in the United States is estimated to contain an equilibrium lung dose of 16 mg of tobacco tar. The Surgeon General asserts that active smoking is a major cause of cancers of many organs of the body, is causally related to coronary heart disease, and is a leading contributory factor in respiratory disease mortality. Since there is no safe level of consumption, the modeled doses from passive smoking, being well within the range of exposure of active smoking, suggest that there is a good foundation for the belief that indoor air polluted with tobacco smoke poses a significant threat to the health of nonsmokers. (Auth. Abs. Mod.)

84–1499. Rodriguez, I.; Santana, J. C.; Sanabria, J.; Banoczy, J. **Prevalence of Oral Leukoplakia in Havana City, Cuba.** *Community Dentistry and Oral Epidemiology* 11(6): 379-383, December 1983.

Survey data on 749 randomly selected Cuban workers (394 females, 355 males, 50 percent smokers) found that oral leukoplakia and preleukoplakia incidence was 16 times higher in smokers than in nonsmokers. Lesions were diagnosed in 33 of the workers (20 cigarette smokers, 3 cigar smokers, and 8 who used cigarettes and pipe). Only two diagnosed patients were not smokers. Smoking for 11 to 40 years was characteristic of the highest incidence. For all disorders, incidence was three to four times higher in males than in females. Findings support previous reports of a strong relationship between smoking and oral leukoplakia in Latin Americans. Areas mainly affected were buccal mucosa and labial commissures. A high involvement of the tongue was not observed.

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

84–1500. Barrett, J. E. **Interrelationships Between Behavior and Pharmacology as Factors Determining the Effects of Nicotine.** *Pharmacology Biochemistry and Behavior* 19(6): 1027-1029, December 1983.

Nicotine and electric shock, unlike many commonly used drugs, can function either as reinforcers capable of maintaining behavior or as punishers that suppress behavior. The reinforcing or punishing properties of consequent events depend on the prior experience of the organism and on conditions existing at the time the events occur. Under some conditions, nicotine and electric shock are able to maintain high levels of schedule-appropriate responding when they are response produced, but will maintain responding when postponed and suppress responding when produced under other circumstances. The multiple behavioral properties are created by and emphasize factors other than physical and molecular features. Studies showing that nicotine and events such as shock can produce dual behavioral effects have drawn attention away from classes of events with categorically restrictive boundaries. The interrelationships between behavioral and pharmacological factors affecting the action of drugs such as nicotine, and other environmental influences on behavior, are thus better understood.

84–1501. Bowers, E. J.; Allen, I. E.; Hickey, R. J.; Miller, G. H. **Cigarette Smoking: Example of Behavioral Regulation of Physiological Homeostasis? (Letters).** *Public Health Reports* 99(2): 108-110, March-April 1984.

A recent study (*Public Health Reports* 98(4): 343-349, July-August 1983) that attributed the difference in longevity between men and women to smoking is criticized. It is proposed instead that smoking (nicotine administration) is symptomatic of a physiological need in some individuals, similar to the need for insulin in diabetics. Nicotine causes the release of monamine neurotransmitter hormones such as epinephrine, which has a therapeutic effect in ulcerative colitis. Also, nearly all female mammals and females of nonsmoking human populations live longer than their male counterparts. Further, the MRFIT trial failed to show any benefits from reduced smoking in the study population. In reply, the author states that the critique implies smokers are constitutionally different than nonsmokers, which is contrary to epidemiological evidence, and adds that final analysis of the MRFIT data showed a twofold decrease in cardiovascular disease risk. The claim that humans engage in smoking to counteract a relatively rare and nonfatal disease while incurring risk of death from other diseases is considered unrealistic. See also 84-0082.

84–1502. Bruno, J. E.; Doscher, L. **Patterns of Drug Use Among Mexican-American Potential School Dropouts.** In: Eiseman, S.; Wingard, J. A.; Huba, G. J. (Editors). *Drug Abuse. Foundation for a Psychosocial Approach.* Farmingdale, New York, Baywood Publishing Company, Inc., 1984, pp. 152-161.

Mexican-American high school students identified as potential dropouts were found to be frequent users of cigarettes and other social drug substances. A survey of 78 potential dropouts in 1975 revealed that 50 used cigarettes daily, 8 smoked several times/week, and 5 smoked at least once/week. The proportion of ever-smokers (67 percent) was similar to that reporting use of alcohol at any time (79 percent for beer/wine, 64 percent for hard liquor) and ever use of marihuana (67 percent). Changes over the past year showed that 56 percent of the surveyed students increased cigarette consumption. Student use of other drugs ranged from 4 percent for hallucinogens other than LSD or opiates to 32 percent for amphetamines; 11 percent used LSD or cocaine, 20 percent sniffed various materials, and 22 percent used barbiturates. Because it is difficult to obtain data from these students (who are often absent or uninterested in school activities), and since participation in the study was voluntary, it is suggested that drug use may be even higher among those potential dropouts who chose not to participate.

More research is needed in the areas of drug use of minority students and its relationship with school performance, and it is suggested that drug education programs need to be directed toward the student who is chronically absent or truant.

84–1503. Brunswick, A. F.; Messeri, P. **Causal Factors in Onset of Adolescents' Cigarette Smoking: A Prospective Study of Urban Black Youth.** In: Shaffer, H.; Stimmel, B. (Editors). *The Addictive Behaviors.* Advances in Alcohol and Substance Abuse, Volume 3(1-2). New York, Haworth Press, 1983-1984, pp. 35-52.

A longitudinal analysis of long-range influences of smoking behavior was conducted among 536 urban black adolescents. Six to eight years intervened between measurement of predictors and subsequent smoking initiation. Separate prediction models were tested for males and females, based on a multivariate causal model that spanned five domains of prior conditions: personal background, school achievement, family—peer orientations, psychogenic orientations, and health attitudes and behaviors. Although variables from all five domains influenced subsequent smoking, different predictors were implicated for adolescent males vs. females, supporting a major hypothesis: salient experiences governing the decision to start smoking are different for black males and females. Applying the same prediction model to cigarette smoking and to illicit drug involvement showed no overlap in prediction for males and but two of the same influences to be significantly implicated in females' illicit drug use and cigarette smoking. (Auth. Abs. Mod.)

84–1504. Burr, R. G. **Smoking Among U.S. Navy Enlisted Men: Some Contributing Factors.** *Psychological Reports* 54(1): 287-294, February 1984.

Factors contributing to the high incidence of smoking in a sample of 505 Navy enlisted men were examined, using Levenson's 1973 multidimensional locus of control scale to assess personality differences between smokers and nonsmokers. Perceptions of stress and peer relationships were also measured. It was hypothesized that nonsmokers would be more internal, less chance oriented, report less stress, and perceive less positive peer relationships than smokers. The results generally supported the first three hypotheses, but not the fourth. It is suggested that programs to promote internal expectations, such as the Navy's Health Readiness Program, could be a method for lowering the high incidence of smoking. Because the proportion of variance that could be accounted for was low, more immediate and salient solutions may be to (1) encourage those in the chain of command to set an example by not smoking, (2) stop the practice of giving work breaks for smoking, and (3) discontinue military price discounts on cigarettes. (Auth. Abs. Mod.)

84–1505. Centers for Disease Control. **Behavioral Risk-Factor Prevalence Survey—United States, Fourth Quarter 1982.** *Morbidity and Mortality Weekly Report* 33(18): 247-249, May 11, 1984.

Data from a behavioral risk factor survey were used to determine the prevalence of smoking in Alaska, California, Illinois, Pennsylvania, South Carolina, and Wyoming. Information was collected through random-digit-dialing telephone surveys in all States but South Carolina (person-to-person/household interviews), and grouped according to sex and age (18 to 34, 35 to 54, ≥ 55, and all ages combined). Smoking prevalence was lowest among females over age 55 in South Carolina (16 percent) and highest in males aged 35 to 54 in Wyoming (51.8 percent). Other risk factors studied were obesity, sedentary lifestyle, uncontrolled hypertension, acute heavy drinking, chronic heavy drinking, drinking and driving, and seatbelt use.

84–1506. Chronic Diseases in Canada. **Recent Trends in Tobacco Consumption in Canada and Other Countries.** *Chronic Diseases in Canada* 4(4): 52-54, March 1984.

Tabular data on manufactured cigarette consumption per capita in 110 countries show that Canada ranks fourth with per capita consumption of 2,927. Trends in Canadian tobacco consumption, including cigarettes, fine-cut tobacco (used for hand-rolled cigarettes), cigars, pipe tobacco, chewing tobacco, and snuff are compared with those for Finland for the period from 1974 to 1982. Similar comparisons are made for total annual consumption of tobacco. While per capita consumption decreased slightly in Canada, declines were much greater in Finland. Preliminary data for 1983, however, indicate that Canadian tobacco consumption decreased substantially, reversing the previous trend. Much of the decrease can probably be attributed to the price of tobacco products, which increased faster than the rate of inflation.

84–1507. Crow, C. S. **Smoking Areas On School Grounds: Are We Encouraging Teenagers To Smoke?** *Journal of Adolescent Health Care* 5(2): 117-119, April 1984.

More students reported smoking at the high school that provided a designated smoking area than at the school that prohibited smoking, but no significant differences were found between schools in the number of cigarettes smoked/day. To determine whether the availability of a smoking area on school grounds affected the incidence of smoking among teenagers or the number of cigarettes smoked/day, questionnaires were administered in two suburban high schools similar in size and socioeconomic status. Smokers were defined as smoking a minimum of five cigarettes/week. In a sample of 1,731 students, 28 percent reported smoking. Females constituted 58 percent of the smoking population and represented 51.4 percent of questionnaire responders. Findings suggest that provision of a smoking area increases the incidence of teenage smoking and that the continued high rate of smoking among teenage girls is a particular concern. Health professionals, educators, and parents should emphasize a multifaceted approach to the complex problem of adolescent smoking.

84–1508. Ebert, R. V.; McNabb, M. E.; Snow, S. L. **Effect of Nicotine Chewing Gum on Plasma Nicotine Levels of Cigarette Smokers.** *Clinical Pharmacology and Therapeutics* 35(4): 495-498, April 1984.

Subjects appear to have compensated almost completely for the increased intake of nicotine from 2 mg nicotine gum by decreasing inhalation of tobacco smoke. To determine whether the use of nicotine chewing gum modifies smoking patterns, 12 regular cigarette smokers were studied for 4 days. On the first day, subjects smoked as usual, and on the second, third, and fourth days, also chewed either 2 mg or 4 mg nicotine gum. They were instructed to smoke as usual throughout the study. Mean plasma nicotine concentration was 29.5 ng/ml with the placebo gum, 30.9 ng/ml with the 2 mg gum, and 40.7 ng/ml with the 4 mg gum. While the number of cigarettes consumed did not change, the mean peak carbon monoxide level was lower with nicotine gum than with placebo gum. Nicotine compensation provided by the 4 mg nicotine gum was only partial. (Auth. Abs. Mod.)

84–1509. Gaiser, J. **Smoking Among Maoris and Other Minorities in New Zealand.** *World Smoking and Health* 9(1): 7-9, 18, Spring 1984.

Cigarette smoking, which is more prevalent among Maoris than among other population groups in New Zealand, coincides with the incidence of lung cancer deaths. Maori women, in particular, are believed to have one of the highest lung cancer rates in the world. Probable influences on the high smoking rate include the Maori's low social class and resultant loss of self-esteem, culture conflict, and dispossession of land following European colonization. Intervention strategies should consider these factors and should recognize the importance of selecting an educator from the same cultural group, implementing changes slowly, using oral communication techniques, and ensuring involvement of the entire community.

84–1510. Goldberg, S. R.; Spealman, R. D.; Risner, M. E.; Henningfield, J. E. **Control of Behavior by Intravenous Nicotine Injections in Laboratory Animals.** *Pharmacology Biochemistry and Behavior* 19(6): 1011-1020, December 1983.

Recent studies on the control of behavior by nicotine injections in laboratory animals demonstrate that such control can be achieved in different ways depending on the behavioral history of the subject and the schedule of reinforcement under which nicotine is administered. Lever-pressing responses by squirrel monkeys and beagle dogs were maintained well above saline substitution levels by injections of 10 to 30 μg/kg nicotine under fixed-ratio schedules. Lever-pressing responses by squirrel monkeys were also well maintained by injections of 30 to 300 μg/kg nicotine under a fixed-interval schedule. The highest rates of responding were maintained by injections of 10 to 30 μg/kg nicotine under second-order schedules in which responding by monkeys produced brief-light presentations that were only occasionally paired with nicotine injection. Under other conditions, however, response-produced injections of the same doses of nicotine suppressed food-maintained fixed-ratio responding by monkeys during the punishment component of a multiple schedule. Finally, under a schedule of nicotine postponement, injection doses of 30 to 66 μg/kg nicotine maintained responding that prevented, rather than produced, nicotine injections. Findings indicate that nicotine may control smoking behavior in humans in very complex and divergent ways, depending on prevailing environmental conditions. (Auth. Abs.)

84–1511. Greenberg, B. S.; Fernandez-Collado, C.; Graef, D.; Korzenny, F.; Atkin, C. K. **Trends in Use of Alcohol and Other Substances on Television.** In: Eiseman, S.; Wingard, J. A.; Huba, G. J. (Editors). *Drug Abuse. Foundation for a Psychosocial Approach.* Farmingdale, New York, Baywood Publishing Company, Inc., 1984, pp. 187-197.

Analysis of approximately 60 hours of representative commercial network programming indicated that fictional television series reflected tobacco use in 23 percent of episodes during the 1976-1977 season and 12 percent during the 1977-1978 season. Viewers typically had to watch for 2 hours to observe smoking of a cigar, cigarette, or pipe during the latter season, compared with 21 minutes for alcohol consumption. Usage rates of cigarettes, alcohol, and illegal drugs varied according to time of day, day of week, and program type. Overall, Saturday shows averaged less than one act per hour, although frequency after 8:00 p.m. increased significantly. Crime shows were the most consistent purveyors of all three substances, while situation comedies were the least. Findings are examined from the perspective of social learning theory, which suggests that exposure to content stimuli that are consistent in theme and occur with considerable frequency can have significant effects on the attitudes and behavior of young viewers. Additional attributes of alcohol users (sex, race, age, socioeconomic status, and role played) are listed.

84–1512. Hay, D. R. **Smoking and Health — New Zealand Statistics, 1984.** National Heart Foundation of New Zealand, Technical Report Series: To Medical and Allied Professions, Report No. 40, Auckland, February 1984, 4 pp.

Important statistical information concerning smoking in New Zealand is summarized as a reference source for doctors and other health workers. Census data for 1981 showed 35 percent of males and 29 percent of females were regular smokers compared with 40 and 32 percent, respectively, in 1976. For comparison, figures for selected countries are provided. Smoking rates by ethnic group show Maoris had the highest prevalence; 54 percent of the men and 58 percent of the women were smokers. However, average daily consumption among Maoris has declined. Statistics also show smoking rates by occupation (highest rates were among the unemployed, 56 percent of males and 52 percent of females); mortality attributable to smoking; lung cancer mortality in 1982 (75 percent increase in women since 1970-1972); and adolescent smoking trends. Factors involved in initiation of smoking are listed, and problems related to smoking during pregnancy and passive smoking are discussed briefly. It is noted that, according to 1981 census data, 24 percent of men and 14 percent of women (424,563 persons) are ex-smokers.

84–1513. Hay, D. R. **Intercensal Trends in Cigarette Smoking in New Zealand 1: Age, Sex and Ethnic Status.** *New Zealand Medical Journal* 97(755): 283-285, May 9, 1984.

A significant change in smoking behavior has occurred in New Zealand in a relatively short period and without a major effort by health authorities. Between 1976 and 1981, the proportion of cigarette smokers declined from 40 to 35 percent among males and from 32 to 29 percent among females. In men, there has been a reduction in every age group, but in women, smoking has increased in those aged 20 to 24 and 70 to 74. More girls than boys aged 15 to 19 are smokers. Smoking rates among Maoris are much higher than in other ethnic groups, but since 1976 there has been a considerable reduction in the proportion of smokers. An important exception is smoking by Maori women 20 to 24 years of age, which has increased to a very high level (70 percent). Almost half of the men and a third of the women who smoke consume ≥ 20 cigarettes/day; however, the average number of cigarettes smoked/day has fallen since the 1976 census to 18 for men and 14 for women. The reduction in smoking has, to a large degree, resulted from increased numbers who have given up the habit, although there are also encouraging signs that fewer boys may be starting to smoke. (Auth. Abs. Mod.)

84–1514. Hay, D. R. **Intercensal Trends in Cigarette Smoking by New Zealand Doctors and Nurses.** *New Zealand Medical Journal* 97(574): 253-255, April 25, 1984.

In New Zealand, the medical profession continues to lead the way in renouncing the smoking habit. If younger doctors maintain the present strong trend against smoking, it is not unreasonable to expect a generation of nonsmoking doctors by the year 2000. The proportion of smokers has fallen in male doctors from 20 percent in 1976 to 15 percent in 1981 and in female doctors from 17 to 13 percent. The same trends have occurred among nurses; a reduction from 49 to 39 percent in men and from 36 to 31 percent in women. However, smoking rates remain high among female psychiatric nurses (46 percent) and male general nurses (48 percent). Only 10 percent of doctors under 24 years of age are smokers. Among general practitioners, the proportion of smokers has fallen from 21 to 14 percent, and among surgeons, from 22 to 13 percent. In a 1983 report on coronary heart disease, the National Heart Foundation of New Zealand outlined strategies for the prevention of coronary heart disease that included "a realistic goal" to reduce smoking from the present 30 percent to 20 percent in a decade. Various professional groups including doctors have already achieved this and shown that it is possible. The challenge is to develop the sort of smoking control programs that will enable other less advantaged social groups to achieve the same goal. (Auth. Abs. Mod.)

84–1515. Henningfield, J. E.; Goldberg, S. R. **Control of Behavior by Intravenous Nicotine Injections in Human Subjects.** *Pharmacology Biochemistry and Behavior* 19(6): 1021-1026, December 1983.

Results, from studies in which procedures used to assess the reinforcing and aversive properties of drugs in animals were extended to a human paradigm, show that nicotine produced the same constellation of stimulus properties whether functioning as a positively or negatively reinforcing event. Smokers (six males, aged 21 to 50, with drug use history ranging from light drinking to polydrug abuse and dependence) were tested using drug self-administration and avoidance procedures, whereby pressing a lever under a fixed-ratio schedule resulted either in the intravenous i.v. injection of nicotine or in the avoidance of programmed i.v. injections of nicotine, respectively. Nicotine was found to maintain responding that produced its injection under certain conditions and to maintain responding that avoided its injection under other conditions. These functional properties of nicotine may be determined by schedule of access to nicotine, dose of nicotine, and past history of the subject. The authors summarize results of studies on nicotine self-administration in animals and humans on pages 989-992 of the same issue of *Pharmacology Biochemistry and Behavior.* (Auth. Abs. Mod.)

84–1516. Henningfield, J. E.; Goldberg, S. R. **Nicotine as a Reinforcer in Human Subjects and Laboratory Animals.** *Pharmacology Biochemistry and Behavior* 19(6): 989-992, December 1983.

Results are summarized from 17 studies in which intravenous nicotine was evaluated in self-administration paradigms. Six species, ranging from the albino rat to the human, have been tested under a variety of schedules of reinforcement and as a function of several pharmacologic manipulations. Under certain environmental conditions, it is clear that nicotine can serve as a reinforcer. However, nicotine differs from many other drugs of abuse in that the range of environmental conditions under which it serves as a reinforcer appears to be more restricted. (Auth. Abs.)

84–1517. Klatsky, A. L.; Siegelaub, A. B.; Landy, C.; Friedman, G. D. **Racial Patterns of Alcoholic Beverage Use.** *Alcoholism: Clinical and Experimental Research* 7(4): 372-377, Fall 1983.

There was little difference between the sexes in proportions who reported current cigarette use among 59,766 persons who had routine health examinations in the years 1978 through

1980, classified by race, alcohol consumption, and smoking status. A powerful drinking-smoking association was evident in both sexes, apparently stronger in women than in men at the upper drinking levels. Past drinkers were two to three times as likely as lifelong abstainers to report smoking and did so in roughly the same proportion as current drinkers. The strong drinking-smoking relationship was present in all race-sex subgroups. Among black males, only 19.1 percent of current smokers do not drink, compared with 43.5 percent who do. For black women, the proportions are 15.7 and 43.3 percent, respectively. Although not as strong, a similar relationship with alcohol consumption is also found for smokers listed as white, Chinese, Japanese, Filipino, or Latin. The proportion of smokers has declined in both sexes in the past 15 years, but the drinking-smoking association has not changed appreciably.

84–1518. Kunze, M. **Smoking Trends in Europe.** *European Journal of Respiratory Diseases* 64(Supplement 126): 103-106, 1983.

Cigarette consumption trends in the European community are discussed, with more detailed information provided for the United Kingdom, the Scandinavian countries, Spain, and Austria and for physicians. In most countries, cigarette sales are declining, with smokers now in the minority (usually less than 40 percent of the population). The exceptions are Hungary and Spain, which have only recently attempted antismoking activities. It is noted that 45 percent of physicians in Hungary are smokers, and in Spain, 58 percent of adult males, 20 percent of adult females, 62 percent of 16- to 24-year-olds, and 75 percent of children aged 12 to 15 smoke cigarettes. Austria has conducted the most intensive mass media campaign, and the Scandinavian countries have shown the most appropriate political initiatives. The Germanic countries face the biggest problem of motivating political and public health officials to take a stand against tobacco products, and the challenge in Great Britain is to maintain the positive momentum reflected by declining consumption.

84–1519. Lancet. **Cigarette Sales to Children.** *Lancet* 2(8360): 1209, November 19, 1983.

Data from a British survey of tobacconists showed that 91 out of 100 sold cigarettes to children aged 10 to 12. Although it is illegal to sell cigarettes to anyone under the age of 16, and 72 of 96 were aware of the law, over 50 percent claimed they did not sell to children (clearly not what actually happened). Some district medical officers have sent letters to all local tobacconists, but the public, particularly parents and teachers, must demonstrate concern also, by being more vigilant and actively discouraging such sales to children.

84–1520. Lancet. **Why Women Smoke Cigarettes.** *Lancet* 2(8361): 1263, November 26, 1983.

The aggressive advertising of cigarettes targeted specifically at women is suggested as one of the main factors in the continuing high percentage of British women who smoke compared with men. Manufacturers identified women as a susceptible sector of the market, and have perpetuated the myths that smoking keeps one thin or makes one more alluring and sophisticated. With the exception of *Good Housekeeping* (which refuses tobacco advertisements), women's magazines carry an average of 12 pages of ads every issue. Thus, tobacco promotion provides a sizable portion of revenue and little can be done legally to restrain the practice. As one journal stated, if it is legal to sell a product, then it is also lawful to advertise it, which sums up the weak position of health educationists. It is also noted that a number of members of Parliament have vested interests in the tobacco industry.

84–1521. Macrae, F. A.; Hill, D. J.; St. John, D.J.B.; Ambikapathy, A.; Garner, J. F.; Ballarat General Practitioner Research Group. **Predicting Colon Cancer Screening Behavior From Health Beliefs.** *Preventive Medicine* 13(1): 115-126, January 1984.

Compliance in returning a Hemoccult II test for colon cancer was negatively associated with smoking among 581 people (33 percent male, 67 percent female) who visited general practitioners and completed a questionnaire (mainly on health beliefs). Patients were then offered the fecal occult blood test free of charge. Of the 86 percent that accepted the offer and took the Hemoccult kit home, 51 percent complied (i.e., returned specimens for laboratory testing). Almost as many acceptors (26 percent) as refusers (27 percent) were smokers. Among the acceptors, however, twice as many noncompliers (34 percent) as compliers (17 percent) were cigarette smokers ($p<0.001$). Thus, the Health Belief Model components that were predictive of initial acceptance of the test offer differed from components predictive of ultimate compliance. The influence upon compliance of family history of colorectal cancer, history of colonic symptoms, smoking habits, perceived attitudes of spouses, attitude toward detection tests, and subjective stress related to the threat of bowel cancer were also investigated. (Auth. Abs. Mod.)

84–1522. Milkman, H.; Weiner, S. E.; Sunderwirth, S. **Addiction Relapse.** In: Shaffer, H.; Stimmel, B. (Editors). *The Addictive Behaviors.* Advances in Alcohol and Substance Abuse, Volume 3 (1-2). New York, Haworth Press, 1983-1984, pp. 119-134.

The phenomenon of addiction relapse is examined from the "addictive processes" frame of reference, citing studies from the areas of smoking, compulsive drug or alcohol use, and criminal recidivism. The consistently high relapse rates (75 to 90 percent) reported for a variety of compulsive behaviors support the concept of addiction as a family of social problem behaviors that share essential similarities. Biochemical, psychological, and social mechanisms are explored as causal factors that underlie the apparent resistance typical of a wide spectrum of addictive disorders. For example, studies of relapse in smokers show that the presence of other smokers and situations perceived as "somewhat stressful" played an important role in precipitating relapse. Also, nearly one-fifth of subjects had been consuming alcohol prior to relapse, suggesting the possibility of pharmacological interactions. It is further noted that "negative affect" smokers, who smoke to reduce tension, suffer significantly higher relapse rates. A relapse prevention strategy, based on the flexible use of a generic cognitive-behavior modification protocol, is suggested.

84–1523. New Scientist. **Curry-Eating Teenage Smokers Cough On.** *New Scientist* 98(1354): 136, April 21, 1983.

Smoking among 16- to 19-year-olds in Britain has dropped by about 15 percent over the past decade, but 17 percent smoke one cigarette or more/week. Regular smoking was reported by 1 percent of 10-year-olds, 30 percent of 15-year-olds, and 28 percent of males and 36 percent of females among those aged 19. Most experimentation occurs among those aged 9 to 12, the prime target group for antismoking campaigns. After age 12, smoking prevalence in girls exceeds that in boys, and it is noted that 23 percent believe that smoking keeps weight down. Only 8 percent of the regular smokers thought their father would disapprove and only 2 percent felt friends would disapprove, compared with 41 and 26 percent, respectively, who felt their father and friends would not mind. Nonsmokers prefer apples and classical and pop music, and drink less alcohol, while regular smokers like rock and reggae music, go more frequently to discos, and cite curry as their favorite food.

84–1524. Newman, I. M. **Capturing the Energy of Peer Pressure: Insights From a Longitudinal Study of Adolescent Cigarette Smoking.** *Journal of School Health* 54(4): 146-148, April 1984.

The role of adolescent peer pressure in the acquisition and maintenance of the smoking habit was examined in a 13-year longitudinal study of 80 students (40 smokers and 40 nonsmokers). Subjects, interviewed in grades 9 and 11, were followed up 10 years later. Peer pressure was reported in terms of meeting certain desirable image characteristics, not in terms of direct pressure to smoke. Smoking was one of many ways to create an image of being mature, independent, and fun-loving and to receive recognition. By employing the principles of social learning theory, educators can (1) incorporate peer pressure in a positive way by considering ways to change the images that energize undesirable behaviors such as smoking; (2) introduce the learner to alternative ways of achieving the image; and (3) alert (immunize) young people to the manipulative functions of image and peer pressure.

84–1525. Newman, I. M.; Gillespie, T. S.; Mohr, P.; Martin, G. L. **Adolescent Alcohol, Marijuana and Tobacco Use in Nebraska.** *Nebraska Medical Journal* 69(4): 92-95, April 1984.

A spring 1982 survey of 4,517 Nebraska students in 7th through 12th grade revealed that 15 percent of the males and 20 percent of the females were regular smokers (at least one cigarette/week). It is noted that the downturn in adolescent smoking prevalence reported in national data is not yet evident in Nebraska; 1980 survey results showed only 13.5 percent of males and 14.0 percent of females were regular smokers. Incidence of smoking increased with age, weekly allowance/income, urbanicity, and number of parents missing from the household. In comparison, 89 percent of the males and 87.5 percent of the females had consumed alcohol (64.7 and 56.6 percent, respectively, within the last month), and 35.4 percent of the males and 32.9 percent of the females had tried marihuana (28.6 and 27.5 percent, respectively, within the past year). Although incidence of both marihuana and tobacco use increased significantly when one or both parents were missing from the household, the use of alcohol was not influenced. Comparison with national patterns of consumption reveals that cigarettes and marihuana are used more frequently in urban areas, while alcohol use is higher in rural areas.

84–1526. Raudsepp, J.; Rahu, M. **Smoking Among Schoolteachers in Estonia 1980.** *Scandinavian Journal of Social Medicine* 12(1): 49-53, 1984.

The belief that smoking is associated with coronary heart disease, lung cancer, chronic bronchitis, and emphysema was more widespread among ex-smokers and nonsmokers compared with current smokers in a survey of Estonian schoolteachers. Questionnaire data on smoking habits and attitudes about the health risks of smoking from 8,347 (82 percent response) indicated that 40 percent of the 1,336 men were current smokers, while 15 percent were ex-smokers. Of the 7,011 women, 11 percent were current smokers and 4 percent ex-smokers. Among men aged below 30, 30-39, 40-49, 50-59, and over 59 years, the percentages of current smokers were 44, 44, 36, 38, and 45, respectively. For women, the corresponding percentages were 20, 15, 7, 6, and 2. There were no differences in smoking rates between Estonian and Russian men. There were more current smokers (13 percent) among Estonian females than among Russian women (5 percent). Most teachers smoked only cigarettes (92 percent of males, 99 percent of females). Two percent of the males and 15 percent of the females smoked only a few cigarettes/month; proportions of heavy smokers (>20 cigarettes/day) were 10 and 1 percent, respectively, for men and women. (Auth. Abs. Mod.)

84–1527. Roed-Petersen, B. **Smoking Habits in East Denmark at October 1979.** *Scandinavian Journal of Social Medicine* 12(1): 39-47, 1984.

An Omnibus Interview Survey of smoking habits, conducted in October 1979 in 2,308 persons living in Denmark east of the Great Belt, found that among 820 teenagers, more had started smoking before the age of 14 than ever before (64 percent of males, 77 percent of females). The multivariate structure of the data was examined via a log linear model to pinpoint sufficient marginals among multicontingency tables. Dependent variables included smoking habits, smoking levels (under or over 16 cigarettes/day), age of onset, inhalation, and smoking status (nonsmoker, ex-smoker, or current smoker). Determinators were sex, age, employment, educational level, gross income, marital status, occupation, and urbanization. Comparison of the data with figures for 1952-1953 showed that smoking had increased for both sexes. Based on the findings, a person-oriented antismoking intervention program mediated by medical and dental practitioners is outlined. Apart from humanitarian aspects, a cost—benefit analysis may indicate a long-term economic saving from the implementation of a successful program. (Auth. Abs. Mod.)

84–1528. Simpson, D. **Trends in Major Risk Factors. Cigarette Smoking.** *Postgraduate Medical Journal* 69(699): 20-25, January 1984.

No British Government has responded adequately or with a sense of urgency to the smoking epidemic or to the demands for tough action that have been heard from the medical profession for over two decades. Nevertheless, trends in smoking show that the fight is being won, even though at a slower rate than if a comprehensive range of smoking control measures were to be enacted. A major risk factor in coronary heart disease in Great Britain is thus being removed. It is the urgent duty of government, politicians, and society as a whole, rather than just health professionals, to ensure that this trend is maintained and improved still further. (Auth. Abs.)

84–1529. Slifer, B. L. **Schedule-Induction of Nicotine Self-Administration.** *Pharmacology Biochemistry and Behavior* 19(6): 1005-1009, December 1983.

Results of recent studies that have demonstrated nicotine self-administration using a procedure of schedule-induction suggest an interaction between environmental factors, such as an intermittent schedule of other reinforcers, and the pharmacological effects of nicotine. Schedule-induced behaviors occur as an adjunct to behavior that is controlled by an intermittent schedule of reinforcement, and are thus not under control of scheduled contingencies. Using schedule-induction procedures, oral, intravenous, and inhaled self-administration of nicotine have been demonstrated in rats, rats and rhesus monkeys, and humans, respectively. Although self-administration of some doses of nicotine occurred without a concurrent schedule of intermittent reinforcement, schedule-induction resulted in responding maintained by lower doses of the drug and much more rapid initiation of self-administration. The interaction between environmental factors and nicotine may be important in understanding the etiology and maintenance of human tobacco use. (Auth. Abs. Mod.)

84–1530. Tewson, J. **Smoking and Schoolchildren. (Letter).** *Journal of the Royal College of General Practitioners* 33(257): 828-829, December 1983.

In 1983, a survey of 256 fourth form students (55 percent boys, 45 percent girls) in Buckinghamshire County, England, showed that 26 percent (equal numbers of boys and girls) smoked one or more cigarettes/week. Of those who smoked, 32 percent smoked regularly before age 12 and 58 percent wanted to quit. Reasons given for not smoking by nonsmokers were health (78 percent), expense (49 percent), dislike of taste (30 percent), parental prohibition (29 percent), and unsociability (28 percent). Among those whose father, mother, or best friend were cigarette smokers, smoking incidence was 38, 36, and 52 percent, respectively, compared with 19, 20, and 10 percent of pupils whose fathers, mothers, or best friends were nonsmokers. Only 10 percent of the 52 students taking "0" level courses were smokers compared with 30 percent smokers among the 204 students taking combined courses.

84–1531. Thurmer, H.; Botten, G.; Kjoernes, U. **Roking; Overvekt og Slanking. [Smoking, Obesity and Weight Reduction.]** *Journal of the Norwegian Medical Association* 104(1): 9-12, 36, January 10, 1984, Norwegian.

Epidemiological studies show that there is an inverse relationship between cigarette smoking and body weight, and that smoking cessation is often followed by weight gain. Weight gain, particularly among women, is a major obstacle to success in quitting, even though the adverse health consequences of regular cigarette smoking exceed those of moderate weight gain. Smoking cessation programs may be more effective if they recognize the opposing effects of weight gain and smoking cessation, the strong pressures brought on by the slim/thin body as an ideal in society, and the importance of nutritional and physical exercise counseling.

84–1532. Webster, C. **Tobacco Smoking Addiction: A Challenge to the National Health Service.** *British Journal of Addiction* 79(1): 7-16, March 1984.

The evolvement of the British antismoking campaign between 1950 and 1962 is examined, noting that tobacco smoking was an entrenched social habit when the British National Health Service (NHS) was formed in 1948, and little has happened to change the situation. Pioneer research on the relationship between smoking and lung cancer provided the first major test in the field of preventive medicine. Sponsored by the British Empire Research Campaign, research was initiated by Sir Ernest Kennaway and extended by the epidemiological work of Doll and Hill in 1950. The inability of the NHS to effectively mobilize and respond to the need for extensive public education on the hazards of smoking was due largely to internal objections within the Cancer Standing Advisory Committee (SAC). Eventually, the issue came before the more prestigious and broadly based Medical SAC and the Central Health Services, as increasing media publicity resulted in persistent parliamentary questioning. The lack of cooperation from officials in the Ministry of Health created additional problems. A change in official attitude followed the 1962 report of the Royal College of Physicians, but even this landmark publication did not mobilize the NHS to adopt smoking and health as a major issue.

84–1533. Wesnes, K.; Warburton, D. M. **The Effects of Cigarettes of Varying Yield on Rapid Information Processing Performance.** *Psychopharmacology* 82(4): 338-342, March 1984.

The effects of four cigarettes with varying tar and nicotine yields on the performance of a rapid information processing task were examined in 25 male smokers. The order of testing was counterbalanced over days using a 5 x 5 Latin Square design. Smoking not only helped prevent the decrease in speed and accuracy that occurred over time in the nonsmoking conditions, but actually improved performance over baseline levels. Furthermore, the greatest improvements were found with the higher nicotine yielding cigarettes. The objectively measured effects of the cigarettes on performance matched subjective evaluations of the effects of cigarettes outside the laboratory. Findings are discussed in relation to other questionnaire studies and a survey of smoking at work. (Auth. Abs. Mod.)

84–1534. Wilkinson, P.; Tylden-Pattenson, L. **Smoking Habits of Nurses Within the Leeds Area Health Authority (Teaching).** *Health Education Journal* 42(4): 109-113, 1984.

A randomized survey of nurses (n=793, response rate 67 percent) found that 32.5 percent considered themselves regular smokers, 51 percent had never smoked, 15 percent were ex-smokers, and 2.5 percent smoked occasionally. Among current smokers, 69 percent had begun before starting their nursing career. The main reason given for smoking was enjoyment, with only 15 percent citing smoking to relieve stress. However, relationships between smoking and different nursing situations were clearly indicated: community nurses had a lower proportion of smokers (23.5 percent) and a higher proportion of ex-smokers (30 percent); 46 percent of those working with cancer patients were regular smokers; 54 percent of the radiotherapy staff who had been nonsmokers prior to starting nursing subsequently became smokers; and on the psychiatric ward, 62.5 percent of the men and 46 percent of the women were smokers. It was also determined that while 94 percent knew that smoking could be harmful, specific knowledge was poor, suggesting that factual information is needed in antismoking education for nurses, in addition to specific information relating to nurses' smoking behavior, and encouragement either to remain a nonsmoker or, for smokers, to quit.

84–1535. Wynder, E. L.; Goodman, M. T.; Hoffmann, D. **Demographic Aspects of the Low-Yield Cigarette: Considerations in the Evaluation of Health Risk.** *Journal of the National Cancer Institute* 72(4): 817-822, April 1984.

Data from a large-scale case—control study of tobacco-related diseases were analyzed to characterize variables associated with cigarette preference (or type of cigarette smoked). Age, sex, race, education, and religion were found to have a strong influence on the choice of cigarette according to tar and nicotine yield. Amount and duration of cigarette smoking were also evaluated by brand history to determine whether tar yield was associated with these variables. Women smoking cigarettes in the low-tar categories tended to smoke fewer cigarettes/day compared with women smoking cigarettes in the higher tar categories. A similar trend was not found for men. As might be expected, only 2 percent of the men and 3 percent of the women over age 40 had smoked low-tar cigarettes (<10 mg/tar) for 10 years or more. (Auth. Abs.)

84–1536. Zabin, L. S. **The Association Between Smoking and Sexual Behavior Among Teens in US Contraceptive Clinics.** *American Journal of Public Health* 74(3): 261-263, March 1984.

Data on 1,174 teenage girls (mean age 16.9 years) attending 32 contraception clinics in the United States showed a strong negative relationship between age of first intercourse and cigarette consumption level. The association is sufficient to reverse the usual correlation between smoking and age during adolescence. The relationship between smoking and early coitus was evident among blacks and whites despite strong racial differentials, suggesting that those who initiate sexual activity in early adolescence demonstrate levels of smoking well beyond the norm, not only for their age groups, but even for adult populations. Preventive, timely contraceptive behavior was also negatively associated with smoking. Therefore, it may be helpful to design and locate interventions in settings to provide information on smoking as well as contraception, because (1) school-age females are now more frequently smokers than males; (2) smoking is particularly hazardous to health when associated with oral contraception; and (3) smoking may be representative of a cluster of behaviors that are especially problematic when initiated outside the normative schedule of such behavior. (Auth. Abs. Mod.)

SMOKING PREVENTION AND INTERVENTION

84–1537. Aghi, M. B.; Grupta, P.; Mehta, F. **Intervention in the Tobacco Habits of Rural Indian Women.** *World Smoking and Health* 9(1): 10-14, Spring 1984.

The women of Kerala and Andhra, in rural India, typically have precancerous lesions on the buccal mucosa or palate or under the tongue from chewing tobacco and/or reverse smoking. The population is totally unaware of the ill effects of tobacco. Following an initial survey and followup phase, an intervention was planned that consisted of stepwise personal interaction, a film on tobacco and oral cancer, posters, newspaper articles, a folk drama, radio programs, and slides shown at the local movie house. At 4-year followup, 25.6 percent of the males and 29.1 percent of the females in Kerala had reduced consumption, while 5.1 and 13.2 percent, respectively, had quit. In Andhra, 34.7 percent of the males and 58.2 percent of the females had reduced consumption, and 14.8 and 16.1 percent, respectively, had quit. Interestingly, more women were able to quit than men, and those in Andhra, where literacy was lower and the population somewhat poorer, showed even higher quit rates. Personal communication was rated most effective in convincing people of the dangers of tobacco, followed by the film and the folk drama.

84–1538. Auth, J. B. **Promoting Nonsmoker Activism.** *USA Today* 112(2462): 52-54, November 1983.

The antismoking campaign, which is gathering momentum in the United States and elsewhere, has created a dilemma for nonsmokers: how to be assertive without being violence inducing. Suggestions are presented to aid nonsmokers who wish to protect their own health by discouraging smokers from exposing them to secondhand smoke. The far-reaching importance of nonsmoker activism is stressed. Social pressures are often implicated in the initiation of smoking among youth, and social disapproval is often cited as a strong motivational force in encouraging smokers to quit smoking or to abstain from doing so near people who are bothered by it. The discomfort and annoyance and the possible health effects caused by passive smoking, especially among persons with allergies, are discussed. (Auth. Abs. Mod.)

84–1539. Belcher, M.; Hodges, D. **Using Expired Carbon Monoxide Levels To Help Smokers Give Up.** *Occupational Health* 35(11): 489-495, November 1983.

A health education exhibit for employees of a large international company used tests of expired carbon monoxide (CO) levels to encourage smokers to quit. Numerous display panels, a life-sized model called "Smoking Sue," and a video film were also featured. Since CO level is a more reliable indicator of inhalation and exposure than consumption or cigarette yield data, it provides a means of categorizing smokers as heavy or light. Cigar and low-tar cigarette smokers often believe they have adopted a less hazardous form of smoking. For those who had CO levels tested during the exhibit, results and an explanation of their significance were provided in written form. It was emphasized that CO levels return to normal approximately 24 hours after cessation of smoking, and visitors were encouraged to abstain for 24 hours and return for retesting. The exhibit was thus an effective means of countering pessimism about the benefits of quitting, while still drawing attention to the visitor's smoking in such a way that he or she was encouraged to stop. Among the 221 people tested, mean CO level was 23.5 ppm. Although women reported lower consumption and preferred low-yield brands, their CO levels did not differ significantly from those of the men (23.6 vs. 25.2).

84–1540. Brady, J. B. **Behavior, the Environment, and the Health of the Individual.** *Preventive Medicine* 12(5): 600-609, September 1983.

Major causes of death and disability in developed countries have changed markedly during the 20th century, and most risk factors associated with disease (such as cigarette smoking) are now behavioral in nature. Accordingly, successful preventive programs must modify the behavior of the individual, since other approaches often fail to achieve long-term abstinence. The most effective programs are multicomponent behavioral interventions involving systematic application of two basic behavioral principles: stimulus control and contingency management. Stimulus control trains the patient to understand the context of his smoking and to identify environmental cues. Procedures can then be implemented to weaken the link between the cues and the act of smoking. In contingency management, rewards are made contingent on progress in the treatment program. Self-monitoring and the helpfulness of a friendly, competitive relationship with another person trying to quit smoking are emphasized. Results with behavioral interventions have been promising, with 61 percent of the patients in one program attaining immediate abstinence and 32 percent remaining abstinent at the end of 1 year (verified by urinary assays for nicotine). The best programs, however, still have a high relapse rate, suggesting the need for new studies of biological factors involved in smoking addiction, such as the release of β-endorphin, vasopressin, and neuropeptides.

84–1541. Durell, J.; Bukoski, W. **Preventing Substance Abuse: The State of the Art.** *Public Health Reports* 99(1): 23-31, January-February 1984.

Research data suggest that young people at the junior and senior high school levels are the most vulnerable to social pressures that lead to experimental and then regular use of cigarettes and other psychoactive substances. Well-designed programs for teenagers in these age groups try to prevent the onset and development of regular use. Primary intervention strategies developed over the past two decades (media campaigns, school drug education programs, and generic programs) are reviewed, and evaluative research is discussed. Apparently many of these programs have not communicated information in a manner that significantly affected the adolescent audience. The most effective approach involves educating youth on the short-term social and health consequences of smoking and teaching the skills necessary to decline the offer of cigarettes by peers. One promising strategy is the macro approach, which focuses on the entire environment and involves dedicated parents and professionals working together to change communities. A second approach employs the concepts of behavioral and social learning theory to offer positive peer role models for teaching young people the social acceptability and desirability of declining a cigarette. Both approaches have been tested in large-scale projects by the National Institute on Drug Abuse.

84–1542. Holstein, E. C.; Deuschle, K. W.; Bosch, S.; Fischer, E.; Rohl, A. N.; Selikoff, I. J. **Port Allegany Asbestos Health Program: A Community Response to a Public Health Problem.** *Public Health Reports* 99(2): 193-199, March-April 1984.

Smoking cessation assistance is included in the Port Allegany Asbestos Health Program (PAAHP), a unique community-run effort to mitigate anticipated health problems resulting from use of amosite asbestos in a Port Allegany glass factory. Cigarette consumption is a critical factor in determining the severity of health risks consequent to asbestos exposure. The program is the result of a cooperative effort by a labor union, a corporation, community health care providers, and a medical school. All persons (n=1,188) employed by the factory between 1964 and 1972 (when an amosite asbestos product was manufactured), and persons in household contact with them, are eligible for the program. Assistance with smoking cessation is a crucial PAAHP service. The American Cancer Society and the American Lung Association have trained participants to be leaders of cessation groups, and clinics have shown short-term success rates of 30 to 50 percent. Other methods are also being developed. Program personnel are particularly interested in whether PAAHP's health education program can augment independent attempts at quitting, since 95 percent of those who stop smoking do so without formal intervention. PAAHP's other services include intensive medical surveillance and health education about asbestos-related risks for both participants and area physicians.

84–1543. Hosokawa, M. C.; Roberts, C. R.; Hansen, P. L. **Health Education in Rural Missouri: Phase II.** *Health Education*, pp. 18-21, March-April 1984.

Data obtained from a mid-project evaluation of efforts in a rural Missouri county to implement a kindergarten through 12th grade health education program indicate a decrease in the reported use of cigarettes. In 1978, about one-third of third grade students had tried cigarette smoking. After the program had been active for 2 years, the number had decreased to about one-fifth. The proportion of sixth graders who smoked remained the same, while the proportion among ninth graders decreased slightly. In contrast, the proportion of sixth and ninth graders who chewed tobacco or used snuff increased markedly, probably because of adult role models, media advertising, and the covert nature of these products that promotes use in the classroom.

84–1544. Koop, C. E. **Julia M. Jones Lecture.** Presented at the Annual Meeting of the American Lung Association, Miami Beach, May 20, 1984, 16 pp.

Although smoking is still the most important preventable cause of coronary heart disease and cancer, per capita cigarette consumption is now below 3,500, the lowest level since 1949. The slow, steady decline in America is attributable to (1) the impact of the prevention message; (2) economics, since smoking confers risk without benefit and the cost of smoking is increasing; and (3) the growing effect of nonsmoking consumers, voters, and taxpayers, combined with the increasing social unacceptability of smoking. Thus a challenge is issued to bring about a smoke-free society by the year 2000. Nonprofit health organizations, independent communities, physicians, and other health care providers are called upon to begin this major health initiative. Individuals are urged to be more aggressive as nonsmokers, to demand smoke-free schools, transportation, and other facilities, and to ask that the film "Death in the West" be shown on television as a public health service. It is noted that the 100th anniversary year of the invention of the cigarette-making machine is an appropriate time to initiate the goal of a smoke-free society.

84–1545. Lammers, J. W.; Kreuter, M. W.; Smith, B. C. **The Effects of the SHCP on Selected Aspects of Decision-Making Among Fifth Graders.** *Health Education*, pp. 14-17, March-April 1984.

Data from the School Health Curriculum Project (SHCP) in 30 Arkansas schools show that 381 fifth grade students in the experimental group reported a wider range of total reasons when considering a hypothetical smoking decision compared with 365 students in the control group. The study was designed to assess the effect of the SHCP on the level of health knowledge and also on a selected cognitive dimension of decisionmaking skills concerning cigarette smoking. There were no significant differences between the groups on pretest scores or on posttest general health knowledge scores. However, analysis of all variables from the decisionmaking questionnaire showed that experimental and control subjects differed significantly in reasons reported for choosing to smoke or not to smoke. All subjects were asked questions about the types of things considered when making a decision regarding smoking, the kinds of things considered most important in making such a decision, and whether or not the decision would be to smoke.

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Experimental subjects reported more health reasons and fewer social reasons for making the decision, and were more apt to say "no" when asked "would you choose to smoke?" It is concluded that the SHCP can affect an important cognitive dimension of the decisionmaking process, and that it motivates students to apply learned health knowledge when thinking about taking health-related actions such as choosing to smoke.

84–1546. Marty, P. J.; Banks, R. L.; McDermott, R. J. **Arnie the Aardvark: An Innovative Cancer Education Program for the Primary Grades.** *Health Education,* pp. 41-42, March-April 1984.

Recent evidence indicates that school-based smoking prevention programs must be initiated before the child is exposed to peer pressure and other influences. In Minnesota, it was decided that efforts should begin in the primary grades and that programs must be simple and informational, yet sufficiently innovative and motivational to maintain interest levels among young children. The Minnesota division of the American Cancer Society has made available a program consisting of an "Aardvark" costume, a film, and a packet of materials including a program script, pamphlets, and a variety of pupil-centered learning activities. The basic informational content of the program (visit from Arnie the Aardvark, the antismoking film, and followup discussion) takes about 15 minutes, with additional learning activities implemented over a period of several days or weeks. Interest on the part of school personnel is high, and positive feedback has been received from teachers, students, and parents.

84–1547. Petosa, R. **Using Behavioral Contracts to Promote Health Behavior Change: Application in a College Level Health Course.** *Health Education,* pp. 22-26, March-April 1984.

The effectiveness of health behavior contracts in promoting health behavior adoption, including not smoking, remains unproved, following assessment in college students enrolled in a personal health class. Following analysis of current lifestyles, the 54 controls and 63 experimental students received information on the health-related consequences of behaviors such as smoking and tobacco use. Modification of smoking behavior was one of four areas covered by the contracts, which also included programs on cardiovascular fitness, weight management, and alcohol consumption. The contract included goals, behavioral objectives, an 8-week record of progress, and evaluation criteria. Of the 63 experimental subjects, 12 chose the smoking modification contract. Self-reported results indicated that eight successfully quit smoking, while four reduced consumption to a mean of 10 cigarettes/day. However, anonymous pretest and posttest assessments showed that contracting had no significant impact on actual health behavior and revealed strong stability in health behaviors studied over the course of the semester. Since self-evaluation reports appear grossly exaggerated, there is a real need for anonymous surveys.

84–1548. Resident and Staff Physician. **Health Update: The Anti-Smoking Campaign. Twenty Years of Mixed Success.** *Resident and Staff Physician* 30(4): 108-109, April 1984.

In the 20 years since the landmark Surgeon General's report on the relationship of smoking and lung cancer, the proportion of Americans who smoke has declined from a high of 42 percent to 33 percent in 1984. However, 53 million persons continue to smoke and the percentage of heavy smokers (>25 cigarettes/day) has increased. The antismoking campaign has helped millions to quit, and the number of high school seniors who smoke dropped from 28.8 percent in 1976 to 21.1 percent in 1982. But the tobacco industry is alive and well, supplying the 600 billion cigarettes smoked each year. The problem of smoking is particularly acute among women; about the same percentage are smoking today as in 1964. In spite of such mixed success, continuing efforts to reduce smoking will help millions to lead healthier lives in the next 20 years.

84–1549. Ross, W. S. **A Nordic "No" to Smoking.** *World Smoking and Health* 9(1): 19-20, 29-32, Spring 1984.

Smoking is generally forbidden in Norway, Sweden, and Finland, except where expressly permitted, and advertisements are banned on television, radio, and billboards and in all Norwegian and Finnish publications. Those ads allowed in Sweden are small and show only the package and a very visible health warning, plus tar, nicotine, and carbon monoxide yields. Sweden and Finland vary the warning labels on cigarette packs, and all cigarettes in Finland are labeled "harmful" or "very harmful." The Nordic antismoking commitment has also spread through voluntary activities such as nonsmoking promotion at sporting clubs, among pregnant women during prenatal care, in community clinics and hospitals, and in the public schools. A nonprofit foundation called "A Nonsmoking Generation" is working to prevent smoking by creating new attitudes, linking nonsmoking to health, success at sports, popularity, and fun. The foundation is using contests and other marketing techniques similar to those employed by the tobacco companies to promote nonsmoking among children. In addition, the Norwegian National Council on Smoking and Health has organized a countrywide volunteer network of teachers, physicians, and nurses to carry the message.

84–1550. Sachs, D.P.L. **Smoking Cessation: What Are Your Referral Options? A Guide to Stop-Smoking Clinics, Self-Help Kits, and Therapy.** *Journal of Respiratory Diseases* 5(3): 49-51, 54, 56-57, March 1984.

Support for a patient's endeavor to stop smoking and for periodic followup are common to all the office-based approaches to smoking cessation described for use by individual medical practitioners. Self-help manuals and kits can be effective if the physician becomes involved in the program, and psychiatric or psychological counseling may work for some patients, as may hypnosis, but selecting the right program for the individual patient is very difficult. There is no evidence that acupuncture is effective. Commercial programs may not actually achieve the high quit rates claimed but may help some persons who otherwise would not make any attempt to quit. One of the more successful commercial programs is SmokEnders, although it has shortcomings in statistical reporting, in defining success rate at followup, and in overall proprietary constraints on divulging methodology. Similar commercial programs include Shick Clinics and Nicotine Withdrawal Clinics. Basic methodologic information on various cessation programs should be made available to the medical profession so that physicians can evaluate a program's suitability for individual patients. (Auth. Abs. Mod.)

84–1551. Sachs, D.P.L. **Treatment of Cigarette Dependency. What American Pulmonary Physicians Do.** *American Review of Respiratory Disease* 129(6): 1010-1013, June 1984.

A sample ($n=588$) of 2,207 actively practicing American pulmonary clinicians attending the 1983 Annual Meeting of the American Thoracic Society was surveyed to determine what they did to help patients stop smoking. Of those pulmonary internists who had never smoked, 99.1 percent recorded the smoking habits of patients and 100 percent advised smokers to stop. In contrast, pulmonary internists who smoked cigarettes were significantly less inclined to do so (84.6 percent and 92.3 percent, respectively ($p<0.001$)). There was also a trend for more internists who were nonsmokers to provide additional services to patients wishing to stop smoking. Of pulmonary internists, 86.8 percent wanted to learn new techniques to help patients stop smoking. Among the few pediatric pulmonary specialists surveyed, 75 percent felt that determining whether or not patients smoked and providing services to help them stop smoking was "not applicable" to their practice. (Auth. Abs. Mod.)

84–1552. Sattaur, O.; Bell, J. **Doctors Lose Propaganda War on Teenage Smoking.** *New Scientist* (1403): 6, March 29, 1984.

Antismoking propaganda has done little to deter British schoolchildren from smoking, according to a recent survey that was supplemented by tests of lung function in 405 secondary school students from a London borough. Lung power was reduced in smokers compared with nonsmokers after 2 or more years of smoking, the degree of impairment increasing with age. In response, the Health Education Council cites the small sample size as the source of discrepancy with national data that shows a slight decrease in smoking levels among early teenage groups. Findings also reinforce the effects of advertising on the onset of smoking in children and teenagers. It is noted that in Sweden, Finland, and Norway, where media promotion of tobacco products has been restricted in an attempt to prevent young people from taking up the habit, smoking among teenagers has decreased, in some groups as much as 50 percent.

See also, **84–1436**

84–1553. Schnur, A. E.; Thier, H. D.; Glantz, S. A. **A Curriculum for "Death in the West."** California Nonsmokers' Rights Foundation and the Risk and Youth: Smoking Project. University of California, Berkeley, 1983, 30 pp.

A self-contained curriculum is presented for upper elementary and junior high school students to supplement viewing of "Death in the West," an antismoking documentary that contrasts the advertising image of the "Marlboro Man" with the reality of six American cowboys dying of cigarette-related illnesses. Produced in England in 1976 and later suppressed by the Philip Morris Company, the film emphasizes the intrinsically false nature of cigarette advertising and attempts to make the Marlboro Man less attractive to young people. The supplementary curriculum, designed to maximize the educational and emotional impact of viewing the documentary, is based on a comprehensive smoking prevention program created and tested by the Risk and Youth: Smoking Project at the University of California, Berkeley. Activities, which require no teacher background in health or science, focus on (1) the negative physiological effects of smoking; (2) the nature of addiction and problems associated with smoking cessation; (3) efforts by the cigarette industry to attract young people to smoking; and (4) the deviousness of that industry's attempts to suppress important information about the health hazards of smoking.

84–1554. Warner, K. E. **Toward a Global Strategy to Combat Smoking: The 5th World Conference on Smoking and Health.** *Journal of Public Health Policy* 5(1): 28-39, March 1984.

The Fifth World Conference on Smoking and Health (Winnipeg, Canada, July 1983) was devoted to dissemination of scientific knowledge and development of political strategies to combat smoking and its associated morbidity and mortality. Three main themes served as focal points: (1) problems of smoking and women; (2) smoking and children; and (3) smoking in developing countries. Sessions were organized under five categories: health consequences of smoking, smoking cessation, public information and education, economic consequences of tobacco, and legislation/political action. In calling for major initiatives, the action-oriented recommendations indicated the magnitude of the smoking and health problem worldwide and the strength of its social, political, legal, and economic barriers. All governments are urged to (1) adopt regular tax increases on cigarettes; (2) ban production and export of cigarettes with tar yields of >20 mg; (3) require health warning labels on tobacco products; and (4) substantially increase antismoking campaigns. Agencies of the United Nations were urged to cease supporting tobacco growing and to initiate programs to develop alternative crops.

SMOKING CESSATION METHODS

84–1555. Cartwright, A.; Fowler, G.; Russell, M.A.H.; Stapleton, J. **Effect of Nicotine Chewing Gum as an Adjunct to General Practitioners' Advice Against Smoking. (Letters).** *British Medical Journal* 288(6411): 145, January 14, 1984.

A previous article (*British Medical Journal* 287(6407): 1782-1785, December 10, 1983) that claimed a doctor using nicotine chewing gum to encourage smoking cessation could produce approximately 38 ex-smokers and save 10 lives/year is disputed. The estimate is considered unrealistic because (1) many patients would already have a smoking-related disease; (2) those who quit would probably be the lighter smokers; (3) the "pool of persuadable smokers" is not inexhaustible; (4) many who quit do not remain abstinent; and (5) many patients seen at later stages are the same ones seen earlier in intervention. It is also questioned how each of the 29,000 general practitioners in the United Kingdom could save 10 lives/year unless the figure of 100,000 smoking-related deaths/year reported by the Royal College of Physicians is an underestimate. The authors reply that the estimates were not intended as detailed epidemiological statistics, and many qualifying factors were omitted. It is noted, however, that the main conclusion of the study, the efficacy of nicotine gum in physician-directed smoking cessation, is not challenged, and a more informed estimate on the number of lives that would be prolonged, and for how many years, is welcomed. See also 84-1252.

84–1556. Gillams, J.; Lewith, G. T.; Machin, D. **Acupuncture and Group Therapy in Stopping Smoking.** *Practitioner* 228(1389): 341-344, March 1984.

Acupuncture and group therapy were compared as methods of smoking withdrawal in 81 smokers who had been smoking >50 cigarettes/week for >5 years. Group A (14 males, 14 females) received acupuncture at a point effective for smoking withdrawal; group B (14 males, 13 females) received acupuncture at an ineffective point; and group C (11 males, 15 females) received only group therapy. At 6 months posttreatment, 18 percent of group A, 15 percent of group B, and 11 percent of group C had given up smoking completely. Although findings indicate that acupuncture is not as effective in helping smokers to quit as has been claimed (at least 50 percent of smokers in group A should have been successful), it is probably as effective as any other single antismoking approach. It is suggested that acupuncture may have a nonspecific effect, which may be physiological, psychological, or both. When administered in any site, acupuncture may cause endorphin release that in turn alleviates symptoms of smoking withdrawal. (Auth. Abs. Mod.)

84–1557. Kramer, J. F. **A One-Year Follow-Up of Participants in a Smoke Stoppers Program.** *Patient Counselling and Health Education* 4(2): 89-94, 1982.

Baseline data from the Smoke Stoppers program show a success rate of 40 percent at best (and no worse than 32 percent), but it is suggested that a more complete preprogram assessment of clients would further define the types of smokers who are likely to succeed. Such an assessment should include knowledge of smoking hazards and measures of attitudes and behaviors in addition to demographic data. The followup study was conducted at least 1 year after participation to determine the effectiveness of the program and to identify characteristics of those who remained nonsmokers. Those who stopped had attended significantly more classes and had a greater proclivity to gain weight than those who resumed smoking. The program, which relies on methods of behavior change, desensitization, and information, consists of four 1.5-hour classes during 1 week, followed by approximately three maintenance sessions. Characteristics not significantly correlated with success were age, education, number of attempts to stop smoking, reasons for wanting to stop, total life stressors, moral support for quitting, companions with whom participants attended classes, number of cigarettes smoked, number of years smoking has continued, and employment with the hospital that ran the program (employees paid reduced fees). (Auth. Abs. Mod.)

84–1558. Murphy, D. H. **Helping Your Patients Stop Smoking: New Resources, New Approaches.** *American Pharmacy* NS24(4): 46-48, April 1984.

The uses and possible benefits of the newly available (January 1984) nicotine gum Nicorette are discussed, citing British trials that found patients who used the gum had higher abstinence rates at 1-year followup compared with users of a placebo gum. The differences in pharmacologic effects compared with inhaled nicotine are discussed, noting the amount absorbed by chewing depends on how hard and long the gum is chewed. Several earlier, less successful cessation methods are reviewed, concluding that virtually all endorse one common element: enthusiastic support from peers and role models, particularly health professionals. The role of the pharmacist is described, suggesting materials and advice that can be supplied without appearing to be judgmental. With the availability of nicotine gum, pharmacists can play an increasingly important role in encouraging cessation. National groups offering antismoking programs to which patients can be referred are listed.

TOBACCO PRODUCT ADDITIVES

84–1559. Boden, R. M. **Branched Ketones, Organoleptic Uses Thereof and Process for Preparing Same.** United States Patent No. 4,380,674, April 19, 1983, 49 pp.

The branched ketone compositions described impart sweet, woody, oriental, camphoraceous, fruity, and spicy aroma and taste nuances to tobacco prior to smoking, and woody, peppery, and oriental-like taste and aroma nuances upon smoking. Recommended concentrations are from 50 to 1,500 ppm, but up to 15,000 ppm is satisfactory. The compounds are prepared by reacting dimers of isoamylene with acyl halides or acyl anhydrides or mixed acyl anhydrides.

84–1560. Boden, R. M. **Branched Chain Olefinic Alcohols, Thiols, Esters and Ethers, Organoleptic Uses Thereof, Processes for Preparing Same and Intermediates Thereof.** United States Patent No. 4,380,500, April 19, 1983, 58 pp.

The uses as a tobacco flavorant of branched chain olefinic tertiary alcohols are described. In various formulations, the compounds provide amber, woody, camphoraceous, floral, orris-like, and fruity aroma profiles with patchouli-like top-notes. A sample formulation is given, which when 20 ppm are applied to experimental cigarette tobacco, results in more black tobacco-like, more aromatic, sweet, bitter, richer, less harsh, and more "cigar box-like" smoke flavor and aroma.

84–1561. Cohn, C. C. **Treatment of Cigarette Paper.** United States Patent No. 4,453,553, June 12, 1984, 11 pp.

A fire-resistant cigarette with lower tar and nicotine delivery and reduced sidestream smoke is described that consists of a charge of tobacco in a low-porosity wrapper substantially free of burn accelerator, treated with water or ethyl alcohol. Fire resistance is improved by depositing a linear burn-rate-reducing substance on the paper from the group consisting of citric acid, magnesium citrate, magnesium acetate, tartaric acid, acetic acid, lactic acid, a sugar, nonfat milk, and skim milk. Similar results are achieved with medium- to high-porosity cigarette papers by treating them with a burn-rate-reducing substance. With high-porosity cigarette paper, if only a part of the surface area is treated, the porosity of the untreated areas controls tar, nicotine, and carbon monoxide delivery. Further improvements in sidestream smoke reduction and fire resistance may be achieved by use of an additional layer of high-porosity cigarette paper in the wrapper. (Auth. Abs. Mod.)

84–1562. Mitchell, T. G.; Barnes, A. G.; Jackson, J. S.; Bevan, P. C. **Smoke Flavor Enhancing Agents.** United States Patent No. 4,441,514, April 10, 1984, 5 pp.

Treatment of tobacco with one or a combination of the compounds 3α-hydroxysclareol, 3β-hydroxysclareol, and 3-oxosclareol enhances the smoke flavor characteristically associated with the particular tobacco. Concentrations of from 50 to 2,000 ppm are recommended. A wide range of microorganisms may be used in the transformation of sclareol to the described flavoring products.

84–1563. Owens, W. F. **Wrapper for Smoking Articles and Method.** United States Patent No. 4,450,847, May 29, 1984, 5 pp.

A wrapper for cigarettes, cigars, and similar tobacco products is described, which contains a filler of precipitated amorphous magnesium hydroxide gel and either an unreactive grade of magnesium oxide or calcium carbonate. For effective reduction in sidestream tar and visible sidestream smoke, the basis weight of the paper should be between 30 and 100 g/m^2. Reductions of at least 20 percent sidestream particulate yields are obtained and reductions of 75 percent can be achieved, depending on the combination of magnesium hydroxide gel, other fillers, and chemicals employed in the wrapper. The filler should constitute 30 to 60 percent of the total sheet weight. The precipitated gel should constitute about 5 to 50 percent (preferably 10 to 25 percent) by weight of the total filler. (Auth. Abs. Mod.)

84–1564. Porenski, H. S.; Sachleben, L. R. **Tobacco Paste Cigarette Additive and Cigarette Having Same.** United States Patent No. 4,452,260, June 5, 1984, 6 pp.

A processed expandable tobacco paste as a cigarette additive and a cigarette having the paste are described. The paste, which consists of a slurry of tobacco stems, dust, scrap, and clippings, is puffed with bicarbonate of soda. The cigarette includes a cylindrical tobacco rod, at least one longitudinally extending line of expanded tobacco paste, and wrapping material that compresses the relatively low-density tobacco rod. The paste can contain a flavoring ingredient. The additive is said to provide a cigarette with firmness of tobacco, even tobacco burning, draw resistance, cigarette end stability, and coal retention comparable to conventional cigarettes, but at a lower tobacco density. (Auth. Abs. Mod.)

84–1565. Vora, P. S. **Characteristics and Applications of Licorice Products in Tobacco.** *Tobacco International* 186(9): 13-14, 16, 18-20, April 27, 1984.

Licorice products have been used in American-blend cigarettes as flavoring/sweetening agents, moisturizing/wetting agents, and flavor-harmonizing additives for tobacco products. New licorice products have been developed for ultra-low-tar cigarettes to harmonize the flavor of an increasing amount of low-quality tobacco, reconstituted tobacco, and expanded stems. More than 100 compounds are found in licorice extract; however, licorice root is the only known vegetation that contains a significant amount of glycyrrhizic acid, which determines the quality, flavor, and price of a licorice product. Glycyrrhizic acid is a saponin consisting of an aglycone of glycyrrhetinic acid and a sugar moiety of two glucaronic acid units. Licorice flavor is the result of a complex matrix of various flavor components, making it nearly impossible to synthesize. The content of sucrose-reducing sugars, ash, and heavy metal residues also affects quality and flavor properties in smoking tobacco. Levels of these components in various licorice products are charted. Cultivation, processing, and quality determination measures are also discussed.

TOBACCO MANUFACTURING AND PROCESSING

84–1566. Cartwright, W. F.; Cornell, A. **Treatment for Natural Leaf Tobacco Wrapper.** United States Patent No. 4,452,257, June 5, 1984, 6 pp.

A method for the treatment of natural leaf cigar wrappers to improve resistance to breakage and enhance taste in conditions of low humidity is described. The treatment system comprises (1) a humectant/plasticizer component, (2) a preservative, (3) a water or water—alcohol solvent carrier, and (4) optionally, a polymeric film-forming component selected from the group comprising synthetic or natural gums and protein film-forming compositions. The treatment is expected to enhance cigar shelf life. (Auth. Abs. Mod.)

84–1567. Friedrich, H.; Ruf, C.; Brosy, J. **Tobacco Extractor.** United States Patent No. 4,448,208, May 15, 1984, 4 pp.

A tobacco extractor is described that consists of a V-shaped conduit and a worm shaft conveyor, which provides counter-current washing for removing soluble constituents from cut tobacco. Conduits are mounted on a frame to form an angle of approximately 160°, so that a large part of the extractor is completely filled with the washing liquid. The worm conveyor is mounted in the conduits and coupled at the point of the V with a cadan or universal joint, and driven by a belt and drive shaft so there are no coaxial moving parts. The spirals of the worm conveyor have a variable compressing action on the tobacco, which aids the extraction process. The solvent flows counter to the direction of the tobacco so that the leaf is completely washed. The extractor can process tobacco continuously.

84–1568. New Scientist. **Patents Filed for Filter That "Cheats."** *New Scientist* 100(1382): 327, November 3, 1983.

Patent applications by Brown and Williamson have been filed in London for a filter tip for cigarettes that has four longitudinal grooves covered by a perforated wrapper to allow air to be mixed with smoke to reduce tar levels. The patents are similar to those filed in the United States, but reflect the manufacturer's awareness of the possibility of crushing the filter while smoking; only a portion of the filter has a porous region, to prevent smoke from entering the grooves while providing a flow path for ventilating air under "certain" smoking conditions (which are never defined). The applications suggest that the company plans a launch, perhaps of the "Barclay" brand (with the "Actron" filter), in Great Britain.

84–1569. Porenski, H. S., Jr. **Cigarette Filter.** United States Patent No. 4,446,878, May 8, 1984, 5 pp.

A cigarette filter is described that directs smoke flow in a lateral direction to increase ventilation and dilution. The arrangement includes a porous filter rod, porous or nonporous plug wrap, and several apertures extending into the filter rod to divert a portion of smoke laterally outside the filter. The apertures extend toward the central axis at a variety of angles and lengths. A baffle that covers all or part of the mouth end of the filter may be included.

84–1570. Ringrose, A.; Crawley, F.E.H.; Coggins, C.R.E. **An Automatic Device for the Addition of Precise Amounts of Radiolabelled Material to Cigarettes.** *Beitraege zur Tabakforschung International* 12(3): 117-121, February 1984.

A microprocessor-controlled device for spiking cigarettes with variable amounts of radiolabeled material is described. Cigarettes can be spiked at rates in excess of 100/min, compared with the very slow manual methods in existence. Data on the disposition of particles is considered essential in the interpretation of rodent inhalation studies. The most widely used method is the addition of (16,17-^{14}C)dotriacontane into cigarettes. Rodents are exposed using special smoke machines. Determination of radioactivity level in respiratory tissues gives an estimate of the total particulate matter deposited. The deposition of the radiolabeled material is very reproducible with a coefficient of variation of around 3 percent. The device is capable of injecting cigarettes of various physical configurations and of producing variable distributions of the radiolabeled material along the tobacco rod. (Auth. Abs. Mod.)

84–1571. Schneider, W.; Schluter, A.; Seehofer, F. **The Effects of the Properties of Materials in a Cigarette on Filter**

Ventilation. *Beitraege zur Tabakforschung International* 12(3): 123-136, February 1984.

A computer model is described that makes it possible to predict the effects of several cigarette properties on the different degrees of ventilation (total degree of ventilation, degree of ventilation of the tobacco rod, degree of ventilation of the filter) and on the pressure drop. The model is able to predict the degree of ventilation from the properties of the materials (e.g., filter, paper, tobacco) instead of from the properties of the manufactured cigarette, making it unnecessary to manufacture an experimental cigarette each time. Fluctuations of the degree of ventilation can be attributed directly to the factors responsible. Each contribution of individual factors responsible can be determined separately (normally not possible by means of experiments). The model, a component of a computer-aided product design, is a useful tool for quality control. A low level of fluctuations in filter ventilation implies constant properties of the applied materials (such as tipping paper) and a uniform primary process. (Auth. Abs. Mod.)

TOBACCO ECONOMICS

84–1572. Ahmad, M. **Iran: Cigarettes in Short Supply.** *Tobacco Reporter* 111(3): 30-31, 34, March 1984.

Cigarettes remain in short supply in Iran and sell at prices 100 to 300 percent higher on the black market compared with official retail prices. Foreign-made cigarettes are either not available or sell at six to eight times the normal price. Shortages continue despite recent local production increases. The Iranian Tobacco Company (ITC), a domestic monopoly, now runs two shifts/day, producing 60 million cigarettes, representing six brands. ITC also imports and markets two local brands produced abroad to meet the taste of former Winston smokers, since a large factory near Tehran had produced large quantities of this product before the 1979 revolution. The shortage may reach 2 to 2.5 billion pieces/year, giving ITC no other recourse but to import cigarettes. Meanwhile, it has embarked on a plan to achieve self-sufficiency in cigarette production over the next 5 years.

84–1573. Bickers, C. **Tobacco Tomorrow: U.S. Leaf Production.** *Tobacco Reporter* 111(4): 46-48, 50, April 1984.

Consequences of deregulation (elimination of the controls on both price and production) of the U.S. tobacco industry are explored. Four characteristics of the industry would have a special impact: the geographic concentration of quota; the potential for expanded production; the demand for American tobacco in both the domestic and export markets; and tobacco's storability at low relative cost. It is estimated that production would move to areas where cost is lowest, the cost of current output would be reduced by about 30 percent, and ultimately, acreage planted to tobacco would expand tremendously. Price stability is also expected, even in the absence of controls. Also considered is what could happen under two possible programs of deregulation: (1) quota phased out through free transferability, with gradual elimination of price supports; and (2) price freeze continued as inflation gradually reduces the real support price, while the total quota is adjusted so that demand is satisfied at a price slightly above the support price.

84–1574. Blalock, T. C. **Some Tobacco Facts—Whether We Like 'em or Not.** *Tobacco International* 186(6): 34-35, 37, March 16, 1984.

Seven factors expected to affect the future course of the American tobacco industry are (1) political aspects of the supply control/price support system; (2) the realities of having to compete in a world market and having to import more tobacco than is exported; (3) the diminishing quality advantage of American cigarettes; (4) technological advances that allow manufacturers more flexibility in type and amount of tobacco used; (5) the need for tobacco farmers to become more cost conscious; (6) effects of increasing taxes, antismoking campaigns, and the worldwide recession on sales and profits; and (7) the lack of price competitiveness in the world market because, for too long, prices were allowed to escalate faster than those of the rest of the world.

84–1575. Burry, R. D. **Cigarette Firms' Cash Flow Healthy; Taxable, Tax-Exempt Shipments Not So.** *Tobacco International* 186(6): 7-10, 12, 14, 16, 19, 20, March 16, 1984.

Near-term earnings estimates for Philip Morris, R. J. Reynolds, American Brands, and U.S. Tobacco for all product lines, including domestic and foreign tobacco, are presented for 1983, and projections of 1985 earnings are given. In addition, production and shipment trends; cigarette pricing at the producer, wholesaler, and retailer levels; and raw material cost patterns are summarized, using data from a variety of industry and governmental sources. The sharp increase in the value of the dollar relative to other currencies had an unfavorable impact on companies, both in translating foreign earnings into dollars and on sales of domestic products in foreign countries. The 1983 tax hike caused substantial distortions in pricing patterns; however, the combination of continued high levels of profitability despite declining domestic cigarette production has maintained strong cash flow patterns. Overall wholesaler shipments to retailers declined 5.7 percent for the first 12 months of 1983, while domestic shipments moved down with wholesalers, but only 3.5 to 4.0 percent. Tax-exempt shipments by producers declined 15.5 percent. Trends in media expenditures are also discussed.

84–1576. Canadian Family Physician. **"Roll-Your-Own" Tobacco Sales Up.** *Canadian Family Physician* 30: 524, March 1984.

Sales of cigarettes and cigarette tobacco in Canada declined 4 percent in 1983, the largest drop in more than 20 years. However, sales of roll-your-own tobacco increased by 7 percent. It is pointed out that cigarettes hand-rolled from loose tobacco deliver as much as four or five times more tar and carbon monoxide, and three times as much nicotine, as manufactured cigarettes with the same brand names. Health and Welfare Canada has asked the Canadian Tobacco Manufacturers Council to indicate the average tar, nicotine, and carbon monoxide yields on packages and in ads for roll-your-own tobacco. The ministry has also requested that manufacturers lower the average tar yield/cigarette to 12 mg and the average nicotine yield to ≤ 1 mg and, by the end of 1984, to ensure that carbon monoxide levels do not exceed tar delivery for any brand.

84–1577. Cole, J. **U. K. Cigar Trade: Consumers Respond to Competitive Marketing.** *Tobacco Reporter* 111(5): 46-48, May 1984.

Although cigars are generally still considered a luxury or semi-luxury item in Great Britain and sales were affected by the recession and heavy tax increases imposed in 1981, the present level is still well above the 1960 level. Domestic cigars make up 80 percent of the market; 20 percent are imported, primarily from Cuba and Jamaica, Europe and countries of the European Economic Community (EEC), and the United States and Canada, and a small percentage from others. U.S. cigars may constitute up to 15 percent of the total market, but are at a disadvantage since they bear the maximum ad valorem duty, making them relatively expensive. European manufacture will no doubt help the price structure, since supplies from the EEC are exempt from the duty. Problems in distribution remain, but British manufacturers are producing several sizes in a variety of packages to appeal to all cigar smokers. Aggressive advertising is a key to the continued success of cigar makers.

84–1578. Dokic, D. **Yugoslavia. Total '82 Production Largest Since '76; Dark Leaf Exports to U.S. Up Greatly.** *Tobacco International* 186(8): 15-16, April 13, 1984.

Because of favorable weather and better agricultural conditions, tobacco production in Yugoslavia in 1982 reached 87,030 tons (59,224 ha planted), an increase of 29.93 percent in quantity and 22 percent in area. This level is close to the record production of 91,412 tons in 1976. Production in the Socialist Republic of Macedonia, one of the most important producing regions in the country, was 36,221 tons (24,682 ha planted), an increase of 16.5 percent in quantity and 22.4 percent in area planted. Besides weather, higher prices encouraged farmers to increase the area planted, contributing to the large crop. Average prices were 18.5 percent higher than in 1981. Exports from Yugoslavia increased 30 percent to 24,918 tons; exports from Macedonia attained 15,971 tons (41.1 percent increase). Exports to the United States jumped 175.9 percent to 6,897 tons. Cigarette exports, however, remained steady at 12 billion pieces (Macedonia exported 2,258 million pieces). Cigarette production was hampered by shortages of equipment parts, paper, cellophane, and packaging material. Macedonia is taking steps to reduce its dependence on imports and to expand production.

84–1579. Gooch, P. **Spotlight on Bulgaria: Setting Sights on Light Leaf Market.** *Tobacco Reporter* 111(3): 64-65, March 1984.

In Bulgaria (one of the largest leaf producers and cigarette manufacturers in the world), most exports go to member countries in the Union for Mutual Economic Assistance, with the Soviet Union the major market. Traditionally a producer of oriental leaf, the Bulgarian tobacco industry is focusing attention on light leaf production, especially Burley, which requires less labor and is more marketable outside the country. Although total tobacco production in 1983 was down 13 percent, Burley yields were nearly unchanged, and area planted to flue-cured and Burley is increasing significantly. Area devoted to oriental leaf is down. Improvement in yields, especially for Burley, are expected, with a goal of 3,000 kg/ha. Availability of raw materials and improved technology have helped the cigarette industry, which has introduced several new brands. Every tenth smoker in the Soviet Union and every fourth smoker in East Germany smokes Bulgarian cigarettes. Although an antismoking campaign has been implemented, it has had little effect on cigarette consumption, which rose from 12,468 billion pieces in 1981 to 13,982 billion in 1982.

84–1580. Gooch, P. **Spotlight on Dominican Republic: Preparing for Self-Sufficiency.** *Tobacco Reporter* 111(4): 38-40, 42, 44, April 1984.

The Dominican Republic, responding to changes in world consumption patterns by switching to light leaf production, is focusing on Burley, which they would like to export after filling domestic demands (imports of light leaf were banned effective January 1, 1984). Light tobacco cigarettes occupy 77 percent of the domestic cigarette market. About 360 acres usually planted to dark leaf are now growing Burley, which could bring total yield to 200,000 metric tons. The dark leaf industry is suffering from both declining markets and increasing costs to growers and packers. To improve cultural practices, the Institute of Tobacco has initiated a new assistance program to provide seeds, fertilizers, pesticides, marketing, technical and managerial assistance, and help in obtaining credit. In the past 6 years, the Republic has emerged as an important supplier of hand-rolled cigars, with 45 million exported last year. Also, the Caribbean Basin Initiative, recently enacted by the United

States, will make it easier for the Republic to compete with Honduran and Mexican cigar filler leaf. Specific cigar and cigarette manufacturers, competition on the international market, and production of traditional tobacco types are discussed.

84–1581. Gooch, P. **Oriental Leaf Report: Surplus Stocks Distort Market.** *Tobacco Reporter* 111(5): 39-42, May 1984.

Demand for oriental tobacco should continue to rise slowly over the next few years as smokers in more of the developing countries change to better quality blended cigarettes. Currently, however, the world supply continues to outweigh demand, particularly in lower grade levels. Wet weather has affected production and leaf quality in Turkey and Greece, the two major exporters, and large stocks of old leaf are a burden. It has even been suggested that the most sensible solution would be to destroy the surplus of old leaf to save carrying charges. Elimination of the export levy in Turkey will probably result in sharper competition and possibly less interest in the 1983 oriental tobacco crop in Greece. To assure a healthy market in Greece, it will be necessary to eliminate stocks of lower grade, abandon cultivation of undesirable varieties, and encourage production of high demand tobacco. The status of oriental leaf production is also summarized for the following countries: (1) Italy, where oriental leaf constitutes approximately 19 percent of total output; (2) Bulgaria, where oriental production has decreased in favor of flue-cured and Burley; (3) the Soviet Union, where weather was generally favorable in 1983; (4) Thailand, where imports from Turkey were up significantly in 1983; (5) the Philippines, where production decreased by 25 percent; and (6) Pakistan.

84–1582. Gooch, P. **Spotlight on Turkey: Excess Rain Reduces Quality.** *Tobacco Reporter* 111(5): 32-33, 36, May 1984.

Poor weather led to high yields of poor quality leaf in 1983 and the challenge for exporters will be to meet demand requirements for American grades without building even larger stockpiles of B grades that may not sell. The forecast for 1984 is much better, and the Turkish Government is seeking to control surplus production by advising farmers not to plant on poor land. The substantial increase in the ceiling price in 1982 motivated farmers to increase output. Tobacco production and area by variety are listed for 1981 through 1983, and export prices for leaf by variety and grade for crop years 1979 through 1981 are given. Exports declined 20 percent in 1982, but because of increased prices, total value decline was less than volume loss. Although production declined in 1982, manufacture of filtered, American-blend cigarettes increased to meet rising demand and manufacturing contracts with Yugoslavia and Bulgaria have been terminated. The tobacco monopoly opposes private manufacture of cigarettes, such as that proposed for the production of Marlboros. Foreign cigarettes, which can now be imported by private companies, still cost about three times more than domestic brands.

84–1583. Grise, V. N. **An Overview of the Tobacco Situation and Outlook for 1984.** *Tobacco International* 186(6): 22-25, March 16, 1984.

U.S. smokers consumed approximately 600 billion cigarettes in 1983, a 5 percent drop from 1982 levels and 20 percent below the 1963 peak, due mainly to the large tax increase and continuing health concerns. Smokers also appear to be returning to full-flavored products, as the market share for low-yield cigarettes fell from 59 percent in 1981 to only 53 percent in 1983. Large cigar consumption declined about 3 percent to 3.57 billion, chewing tobacco sales dropped 2 percent, but snuff use increased 4 percent. Total U.S. tobacco use fell 8 percent to 934.5 million pounds, with cigarettes accounting for 85 percent. Exports dropped 13 percent due to the strong U.S. dollar and domestic use fell 2 percent because of declining cigarette output. Current leaf supply and loan volume, quota status, estimates of production, and related cost increases are detailed. Major changes resulting from recent legislation on the price support and production control program are summarized.

84–1584. Grise, V. N. **Flue-Cured Tobacco Situation and Outlook With the No Net Cost Program.** In: Pugh, C. R. (Editor). *Tobacco Issues for the 80's.* Proceedings of the Conference of the Colleges of Agriculture in the Five Flue-Cured States, March 8-9, 1983, Myrtle Beach, South Carolina. Available from C. R. Pugh, Agricultural Extension Service, North Carolina State University, Raleigh, 1983, pp. 1-6.

Estimates of per capita consumption of cigarettes in the United States indicate that 1982 levels were at a 26-year low (14 percent below the 1963 peak). Demand for flue-cured tobacco depends almost entirely on its use in cigarettes; U.S. blends are about 36 percent flue-cured, and domestic use represents just under half of yearly use. The balance is exported. Because of rising prices, excise taxes, and antismoking activities, consumption is expected to continue to decline. Worldwide production of flue-cured tobacco is increasing, although slowly, and because competing countries can sell at approximately one-half the value of U.S. leaf, exports are declining and are not expected to increase. Under the No Net Cost program, flue-cured growers were assessed 3 cents/pound in 1982 and will be assessed 7 cents/pound in 1983. The legislation also requires corporations, utilities, educational and religious institutions, and other entities owning tobacco allotments but not significantly involved in farming, to sell allotments by December 1, 1983. Because of the supply of cheaper tobacco throughout the world and the problems of allotments and quotas in the United States, a major concern now is what measures can be taken to make U.S. tobacco more competitive in the world market.

84–1585. Hill, D. **Huge Tax Increase Another Blow to British Cigarette Trade.** *Tobacco International* 186(9): 50, April 27, 1984.

Great Britain's new spring budget included an increase of 10 pence on a pack of cigarettes, and taxes now represent about three-fourths of the retail price. An increase of 3 or 4 pence would have been in line with inflation. Tax increases were also announced for hand-rolling leaf and cigars. Although tobacco taxes represent about 5 percent of government revenues, the Chancellor stated that the increases were also generated for health considerations, in spite of warnings by the tobacco industry that thousands of jobs would be endangered. When the duty was increased by 14 pence in the 1981 budget, sales immediately dropped by nearly 20 percent. Following the 1974 peak, cigarette sales have fallen 26 percent, with a current annual decline of about 5 percent. The recent 10 pence increase is expected to be followed by a sales slump of about 10 percent. It is noted that the changing tax structure has also brought about market confusion, with brand proliferation and intense price competition.

84–1586. Houqing, S. **Water-Pipe Tobacco Holds Its Following in China.** *World Tobacco* 84: 111, 113, March 1984.

Although cigarettes are the most popular form of tobacco consumption in the People's Republic of China, some traditional smokers remain loyal to water-pipe tobacco, the specialty of a Lanzhou factory. Total national output of this form of cut or shredded tobacco reached 3.5 million kg in 1983, indicating relative stability in consumption over recent history. Traditionalists continue such smoking because the tobacco has a unique fragrance and is milder and less harmful after being filtered through water, but the cumbersome apparatus is not likely to attract many converts. The tobacco undergoes an elaborate processing during which a number of nontobacco ingredients are added. Common supplements are linseed oil and rapeseed oil to conserve moisture and pliability and to serve as an antiseptic. The tobacco is marketed in two varieties, the green and mian types, with rustica the main ingredient in both varieties.

84–1587. Howland, K. E. **Tobacco Import Restrictions in Major Markets.** In: Pugh, C. R. (Editor). *Tobacco Issues for the 80's.* Proceedings of the Conference of the Colleges of Agriculture in the Five Flue-Cured States, March 8-9, 1983, Myrtle Beach, South Carolina. Available from C. R. Pugh, Agricultural Extension Service, North Carolina State University, Raleigh, 1983, pp. 107-119.

The effect of import restrictions (tariffs and nontariff measures) on world tobacco markets is discussed, noting that the United States remains a comparatively open market for imports, but exports face discrimination in foreign markets. The United States exports unmanufactured tobacco to more than 100 countries, but fewer than 20 take over 90 percent of exports. These same few countries import more than 75 percent of all the leaf moving in world trade. U.S. imports increased from 13 percent to 16 percent of the world total between 1972 and 1981, while exports fell from 23 percent to 18 percent. The United States and most of its trading partners observe rules embodied in the General Agreement on Tariffs and Trade (GATT), under which all members receive treatment equivalent to that accorded the most favored member. Multilateral trade negotiations (sponsored by GATT) have had some success in reducing the level of tariff protections, but less with nontariff restrictions. It is concluded that tariffs probably have little impact on the level or origin of tobacco imports, while nontariff measures and monopoly decisions may be important import determinants. A few major leaf exporting countries, such as Argentina, Brazil, Mexico, and Turkey, maintain high tariffs and therefore have totally protected import markets.

84–1588. Johnson, P. R.; Norton, D. T. **Social Cost of the Tobacco Program Redux.** *American Journal of Agricultural Economics* 65(1): 117-119, February 1983.

Fifteen years ago, it was predicted that (1) the elasticity of foreign demand for tobacco would increase as foreign output increased; (2) institutional rigidities fostering the U.S. monopoly position would erode; and (3) losses due to inefficiency would increase. Current analysis shows that (1) foreign demand elasticity increased, but only a little, and the impact on U.S. exports was smaller than expected; (2) erosion of institutional rigidities is neither confirmed nor denied (market behavior indicates the United States still maintains a quality differential over competing tobacco, aided by the Rhodesian embargo); and (3) inefficiency losses did not grow as expected owing to changes in domestic institutions. It is noted that the inefficiency loss is still relatively large, and a predicted major decline in U.S. market share has been borne out. The surplus loss of foreign consumers that can be ignored in national income accounting is larger than lost domestic surplus. Large monopoly rents derived from foreign sales continue and are sufficient to offset domestic losses. Overall, little has changed in the calculated gains and losses over the 15-year span. It is concluded that the longevity of the U.S. price support program has contributed to this stability.

84–1589. Kiger, H. C. **Outlook for US Flue-Cured Tobacco.** *Tabak Journal International* 1: 6, 8, February 1984.

U. S. flue-cured tobacco, traditionally the most important type used in producing American blend cigarettes, has accounted for 75 to 80 percent of leaf tobacco exports. Changes over the years, however, have clouded the current outlook. Following World War II, the United States dominated world production and trade in flue-cured tobacco; production outside the country was low and of relatively poor quality. However, an increasing number of buyers are now turning away from U. S. flue-cured in favor of less expensive tobacco of comparable quality. The reason for the gradual switch is complex, but analysts believe it can be attributed to the present price support system. Exacerbated by the strong U. S. dollar, the system has priced American flue-cured out of competitive range. As a result, developing countries have increased production and quality of their leaf to meet rising demand. Between 1972 and 1982, average production in China increased 310 percent and in

Brazil 212 percent, while U. S. production showed an 18 percent decrease. The solution is to amend the price support system so that prices are more in line with grower costs and with the competitive realities of the current marketplace. (Auth. Abs. Mod.)

84–1590. Krum, J. R.; Culley, J. D. **Advertising-Campaign Change: Lessons From Leading Cigarette and Liquor Brands.** In: Percy, L.; Woodside, A. G. (Editors). *Advertising and Consumer Psychology.* Lexington, Massachusetts, D.C. Heath and Company, Lexington Books, 1984, pp. 57-73.

Content analysis of hundreds of magazine advertisements for 4 cigarette brands and 14 brands of liquor found that four major product appeals are used: (1) the product itself, (2) product heritage, (3) superior product leadership, and (4) product and its uses. A total of 116 advertising campaigns, which varied in length from 1 to 21 years, were identified. The importance of advertising slogans is discussed, noting that Marlboro's "Marlboro Country" has been used effectively for 19 years; sales increased from 26 billion cigarettes to 110 billion during the period. Sales of Winston, in comparison, increased from 52 billion to only 82 billion utilizing 15 different campaigns. Product-related appeals vs. consumer-related appeals are described, and the value of continuity in campaigns is documented. It is concluded that continuity in advertising campaigns can lead to outstanding results for heavily advertised consumer products such as cigarettes. The essence of great advertising is to create a campaign with long-run appeal and stick with it, but be willing to change if the campaign is not working.

84–1591. Lalande, L. **Seita in the Black Again.** *Tabak Journal International* 1: 26-27, February 1984.

Because of pricing adjustments, grants from the Treasury, more dynamic marketing, and stricter business management, Seita achieved a surplus of 45 million francs in 1982. Losses in 1981 amounted to 165 million francs; annuity contracts that had been drawn up by the former monopoly were taken over by the French Government, and in return, Seita received 200 million francs in 1982. Cigarette sales in France rose 1 percent over 1981 levels to 87.9 thousand million, with Seita's products totaling 58.5 thousand million. The previous 2 years had seen a slight decline in cigarette consumption. Sales of mild cigarettes have increased 9.8 percent over the past few years and now occupy an estimated 92 percent of the market. Foreign trade dropped 4.3 percent, while cigar sales fell 3.4 percent in 1981 and 7.4 percent in 1982. Currently, more aggressive marketing activities are in operation, including the introduction of new brands to cover all segments of the market, and a more dynamic image and increased sports sponsorship for the traditional brands (Gauloises, Gitane, and Royale).

84–1592. Matlick, D. **Spotlight on Spain: Challenging Years Ahead.** *Tobacco Reporter* 111(5): 22-23, 26, 28, 30, May 1984.

In Spain, changes in consumer cigarette preferences, Spain's potential entry into the Economic Community (EC), health issues, contraband, and economic problems in the Canary Islands are bringing about a restructuring of the tobacco industry. An estimated 46 percent of cigarettes consumed in 1984 will be American-blend type compared with 10 percent in 1974. Since 1972, when 4.6 billion blond cigarettes were sold (54 percent imported), demand has increased 20 percent annually, and would have risen faster except for high taxes. Current efforts to resolve tax and procedural differences with the EC will result in even greater demand for blond cigarettes, reduced demand for dark-tobacco products, and an overall tax increase of 8 percent over 1980 levels. The Spanish monopoly, Tabacalera, S.A., is attempting to increase domestic flue-cured leaf production (at the expense of fermented Burley) to match changing consumer preferences, which is raising objections by farmers and causing economic depression in the Canary Islands, where tobacco manufacturing is a major source of income. Tabacalera is also arranging long-term licensing agreements to meet domestic demands and prevent a flood of imports with EC membership. Tabacalera's reorganization plans, agricultural conditions, and conversion of factories for blond cigarette production are also discussed.

84–1593. Maxwell, J. C. Jr. **Cigarette Sales Slide; PM, Lorillard, L and M Gain.** *Tobacco International* 185(26): 40, 42, December 23, 1983.

A 5 to 6 percent drop in cigarette sales by the end of 1983 is predicted (590 to 597 billion units, down from 624 billion in 1982). The doubling of the Federal excise tax and continuing health concerns are cited as the primary adverse factors. Current cigarette consumption appears to be down at least 4 percent at an annual rate. Low tar sales have also topped out, and manufacturers are gradually moving tar content up by several milligrams. Three companies showed absolute volume gains during 1983: Philip Morris was up 0.3 percent; Lorillard gained 1.5 percent; and Liggett and Myers increased 51 percent (because of generic lines, which now have a 3.0 to 3.5 percent market share). Overall, however, the industry shows a realistic outlook, and is focusing on cost control measures, shrinking production schedules, layoffs, and a possible return to price changes only once every 12 to 18 months. Marlboro continues to be the most popular brand, one of the few to increase its market share in 1983 (from 19.2 to 19.9 percent).

84–1594. Peter, R. **Atmosphere of Crisis Among Argentinian Tobacco Growers.** *Tabak Journal International* 1: 9, February 1984.

Tobacco cultivation in Argentina is of great economic importance, especially in the provinces of Salta and Jujuy; large sections of the population depend on it for their living. However, they are faced with extremely serious economic difficulties, largely because of excessively high interest rates on loans and credits, resulting in an enormous increase in the cost of living. Tobacco growers have declared that a reduction in taxes is imperative to enable them to market for export those quantities of tobacco that remain available for this purpose and to enable them to be competitive vis-a-vis other tobacco-exporting countries pursuing a distinctly aggressive and resolute policy. Some reaction to the urgent appeal by the new government headed by Raul Ricardo Alfonsin is anticipated. (Auth. Abs. Mod.)

84–1595. Pilz, C. **Virginia Tobacco is a Bonanza for Philippine Tobacco Farmers.** *Tabak Journal International* 2: 126, 128, April 1984.

The Philippine Virginia Tobacco Association (PVTA) reports crops and exports at record levels during the past 3 years. The February 1983 crop was just under 50 million kilos, an increase of 2.3 million kilos over 1982. Exports were somewhat reduced, but following the 30 percent devaluation of the peso, they are expected to rise rapidly. There is a demand for Philippine Virginia tobacco owing to its low nicotine content, and the PVTA is conducting research and instructing tobacco farmers on how to improve quality. The cultivation of Burley tobacco is also considered promising, as farmers are learning to handle problems relating to the curing of this variety. Cigarette production also increased, from 41 billion pieces in 1978 to 63 billion pieces in 1982. Although the population of 52 million is increasing at a rate of 3 percent, estimates indicate only 20 percent are smokers. (Auth. Abs. Mod.)

84–1596. Pugh, C. R. (Editor). **Tobacco Issues for the 80's.** Proceedings of the Conference of the Colleges of Agriculture in the Five Flue-Cured States, March 8-9, 1983, Myrtle Beach, South Carolina. Available from C. R. Pugh, Agricultural Extension Service, North Carolina State University, Raleigh, 1983, 133 pp.

The conference objective was to provide an information base on tobacco policy issues related to the No Net Cost Tobacco Program, which is having a substantial impact on agriculture in the five southern States producing most of the U.S. flue-cured tobacco crop. Topics discussed included (1) price prospects; (2) implications for rent and the value of quota; (3) implications for the structure of tobacco farms; (4) implications for price stabilization; (5) trends in the quality of U.S. flue-cured tobacco; (6) implications of various technologies to tobacco quality; (7) tobacco marketing issues; (8) industry effects of cigarette tax changes; (9) nature of trade restraints in the world market; (10) world demand for tobacco; and (11) indicators of growth by domestic tobacco companies. The aim was not to foster a particular policy stance but to present objective data that will be useful in assessing policy alternatives. Understanding the issues will help assure the future progress of the industry. See also 84-1584, 84-1587, 84-1600.

84–1597. Rogers, D. **Muddled Thinking?** *Tobacco Reporter* 111(5): 6, May 1984.

The two-sided approach of the British Government in its dealings with the tobacco industry is discussed. The Government pays tribute to the industry's efficiency and innovation in an "extremely difficult situation," but the difficult situation is largely of the Government's own making through high taxes (an average 92 pence of the 1.24 pound cost of a package of cigarettes) and antismoking campaigns, which have led to reduced consumption. In purely economic terms, the tobacco industry is vital to the Government. Behind value added tax and revenue from hydrocarbon oil, tobacco is the third biggest source of consumer revenue. The Government simply cannot afford to tax the industry out of existence, so it pursues a policy of wounding rather than killing.

84–1598. Seuss-Calandrelli, D. **Italian Tobacco: Facts, Figures, Trends. Leaf Tobacco Competitive on Domestic and International Markets.** *Tabak Journal International* 1: 46-48, February 1984.

Between 1971 and 1982, Italian production of leaf tobacco increased 92 percent, from 75,000 to 145,000 metric tons. A more exact selection of varieties, better quality, new cultivation techniques, increased mechanization, and other improvements have increased the share of Italian leaf on the national and international market. Of all Italian tobacco on offer, approximately 15 percent is flue-cured, 45 percent light air-cured, 20 percent oriental, and 20 percent dark, subtropical, and indigenous tobacco. Exports of Italian leaf are rising, while domestic sales of manufactured tobacco products are also increasing. Agricultural production trends for specific varieties and relevant economic conditions are discussed.

84–1599. Seuss-Calandrelli, D. **EEC Tobacco Policy to Be Revised?** *Tabak Journal International* 1: 49-50, 52, February 1984.

Greece's entry into the European Economic Community (EEC) has pushed up expenditures in the tobacco sector to such an extent that the policy of the EEC is being questioned. In addition, predictions on future production from Spain and Portugal have led those concerned to fear the worst. At the Third Eurotobacco Congress in Sorrento, several speakers noted that even after Greece joined the EEC, the Community could cultivate only 45 percent of its needs, and that farmers succeeded in making cultivation profitable only because of present regulations based on price incentives. The basic objectives of the EEC premium system, which are to guarantee selling price, return to the grower, and sale of Community tobacco, have been reached only by West Germany, France, and Belgium, although Italy (the largest tobacco producer) has had some success with its 1981 crop. Nevertheless, EEC economic experts reiterate that sustainment of European tobacco cultivation will strengthen European politics in the long run, and that this is one of the fundamental aims of the agricultural policy of the Community. (Auth. Abs. Mod.)

84–1600. Sheets, T. J. **Trends of Pesticide Residues on Flue-Cured Tobacco.** In: Pugh, C. R. (Editor). *Tobacco Issues for the 80's.* Proceedings of the Conference of the Colleges of Agriculture in the Five Flue-Cured States, March 8-9, 1983, Myrtle Beach, South Carolina. Available from C. R. Pugh, Agricultural Extension Service, North Carolina State University, Raleigh, 1983, pp. 49-51.

Pesticide residues are a factor in determining tobacco quality, and the use of maleic hydrazide (MH) for sucker control has created problems for the U.S. tobacco industry. Average levels are still well above the 80 ppm tolerance proposed by the Federal Republic of Germany, a major importer, although lower than peak levels of about 130 to 140 ppm in the mid to late 1970s. While high MH residues are at present the only known quality problem associated with pesticide use, several other pesticides registered for tobacco must be watched carefully, particularly halogens (chlorides and bromides) that must be applied several times over the season for effective control. Relatively high residues remain on cured leaf, and have been increasing. Data for 1980 show an average of 0.74 percent halogen level compared with 0.58, 0.55, and 0.56 percent in 1974, 1976, and 1978, respectively. A surveillance program must be maintained to detect excessive levels of pesticides, and levels of MH must be reduced by at least 50 percent without deterioration of tobacco quality.

84–1601. Shelton, A. **Tobacco Tomorrow: Cigarette Sales and Markets.** *Tobacco Reporter* 111(4): 54-55, 57, April 1984.

Lingering economic recession, stable smoking populations, tax and price increases, and growing antismoking activity continue to impede growth in the mature cigarette markets of the world. Consumption in most major markets is either static or declining, and the latest available information indicates that world cigarette output showed no growth again in 1983. However, the 4 percent drop in U.S. cigarette production was less than expected in the wake of sales declines due to tax increases and rising prices. Current speculation leans toward a resettling of the market, with more segmentation, renewed or broader emphasis on brand image, and a small niche reserved for economy brands or nonbrand cigarettes. It is anticipated that 1984, at best, will be a break-even year for U.S. cigarette manufacturers. By segment, the market picture suggests a decline in sales of ultra-low-tar brands, slower growth in the low-tar segment, and continued gains in popularity among 100 mm brands. The most serious losses are expected in exports, since European sales were cut severely in 1983 owing to the high level of the dollar and relatively stagnant cigarette market. Manufacturers are concentrating on building new markets in developing countries and on negotiating with nations that have traditionally restricted imports.

84–1602. Shoemaker, R. A. **More Taxes on Tobacco. In River City, or Anywhere Else, Higher Excises Spell Trouble With a Capital T.** *Legislative Policy,* pp. 14-19, January-February 1984.

Faced with rising budget deficits, Federal and State governments often turn to "sin" taxes, especially on tobacco, to raise revenue. However, reduced sales offset the revenue potential of the tax increase. Excise taxes on tobacco are not related to special purposes, as selective taxation should be, and are inflationary because they raise prices while failing to improve the product. In addition to tobacco-related industries, convenience stores and other retail businesses suffer disproportionately from the higher prices caused by tobacco tax increases that also engender interstate bootlegging and tobacco price wars. Low penalties fail to deter illicit cigarette smuggling. User-selective taxes are regressive (especially tobacco excises), affecting fixed-income and lower-income persons the most. Despite also being costly to States, further increases are being considered by the Federal Government, although President Reagan seems opposed to the idea. Raising State excises is not an effective way to compensate for revenue shortfalls, a conclusion shared by many across the political spectrum. (Auth. Abs. Mod.)

84–1603. Steele, I. **Third World Tobacco Growing Scorned in West, Not Third World.** *Tobacco International* 186(4): 7, 11, 13, 17, February 17, 1984.

Third World nations are playing an increasingly prominent role in the world tobacco market. In response to industry critics, a London-based news magazine evaluated the situation in Asia, Africa, Latin America, and the Caribbean, and concluded that tobacco is not responsible for displacing food production and that it contributes significantly to employment and economic productivity in developing countries. The area devoted to tobacco cultivation in the Third World in 1980 was only 0.37 percent of the total arable and permanent crops area, and tobacco land is occupied for only 3 to 6 months of the year. Sponsoring manufacturers and merchants frequently provide tobacco farmers with access to new technologies, and at the national level, the removal of tobacco would result in foreign exchange losses for both exporting countries and those engaged in import substitution. Tobacco farming also benefits small landholders and helps slow urban migration. The World Bank estimates that developing countries (1) will produce 63 percent of world tobacco by 1985 and 67 percent by 1995; (2) consumption will reach 54 and 60 percent, and (3) imports probably will grow to 30 and 33 percent of the world total.

84–1604. Sumner, D. A. **Tobacco Deregulation Would Result in Lower Prices, Increased Output.** *Tobacco International* 186(6): 26-28, March 16, 1984.

Increased foreign tobacco production, antismoking campaigns, and government budgetary problems have given impetus to the tobacco deregulation movement over the past 10 years. The probable effects of abandoning the current tobacco program and allowing development of an essentially unregulated market include (1) elimination of the capital value of tobacco production rights and loss of about 800 million dollars in annual income to quota and allotment owners; (2) a price reduction of 20 to 30 percent; (3) an increase of about 100 percent in the quantity of tobacco exports; (4) substitution of domestic tobacco for most imported leaf used in U.S. cigarette manufacturing; (5) an increase of 25 to 75 percent or more in total revenue to the tobacco production industry; (6) no major problems of price or producer income variability; (7) movement and concentration of the production industry in the least-cost regions; (8) gain in total agricultural and personal income in areas of major tobacco expansion; (9) no increase in monopoly power by tobacco buyers; and (10) the possibility of major costs of dislocation and disruption if price support and reduction restrictions are eliminated suddenly.

84–1605. Tabak Journal International. **Statistics.** *Tabak Journal International* 6: 528-529, December 1983.

Production and import/export statistics for smoking and chewing tobacco and snuff are presented for selected countries worldwide. Production totals are listed yearly for 1978 through 1982, and import/export totals for 1980 through 1982. Changing patterns are reflected in figures for some of the largest producers/importers. In the United States, production increased from 70.8 million kg in 1978 to 73.7 million kg in 1980, exports increased from 8.4 million kg in 1980 to 10.0 million kg in 1982, and imports increased significantly from 14.0 million kg in 1980 to 80.7 million kg in 1982. In the Netherlands, production increased from 24.1 million in 1978 to 31.5 million in 1982, exports increased from 22.7 million kg in 1980 to 23.9 million kg in 1982, and imports decreased from 8.3 million in 1980 to 6.9 million kg in 1982. Production in the Federal Republic of Germany rose from 7.7 million kg in 1978 to 10.5 million kg in 1982, and from 1980 to 1982, exports decreased from 4.1 million kg to 2.5 million kg and imports increased from 18.1 million to 26.1 million kg.

84–1606. Tabak Journal International. **R. J. Reynolds Reorganize International Tobacco Operations.** *Tabak Journal International* 1: 17-18, February 1984.

Because of changing business environments around the world and overall maturing of its own markets, R. J. Reynolds has reorganized its international tobacco business. Activities will be divided into two categories: established markets and undeveloped markets, each under the direction of an executive vice president. Previously, R.J.R. Tobacco International operations were organized along geographical lines, in four units, to be near markets being supported. Under new structure, operations in the existing four areas will be replaced with 10 strategic business units, which will consist of markets with clear business similarities. Each will contain a "core company" headed by a regional vice president who will report directly to the executive vice president. This will streamline reporting and allow local managers more autonomy and flexibility, while providing management more control over strategy and resource allocation. (Auth. Abs. Mod.)

84–1607. Tabak Journal International. **Why Britain's Tobacco Industry Is More Cheerful.** *Tabak Journal International* 1: 14, 16, February 1984.

Although unofficial estimates put 1983 consumption at 2 percent lower than 1982 levels (and 27 percent below the 1973 peak), morale in the tobacco industry is rising, owing in part to the belief that Britain is pulling out of the economic depression. The industry is more efficient than ever following the recent declines in sales, and because of concentration of production in high-performance factories combined with a reduction in excess manpower, manufacturers' profits are improving. The industry is encouraged by a report from the Independent Scientific Committee on Smoking and Health, which concluded that nicotine, at levels derived from smoking, has not been shown to be harmful to the cardiovascular system in healthy people, nor to be carcinogenic or teratogenic. In addition, reduction in tar yields is credited for some of the decline in lung cancer mortality rates. However, the market is still far from serene, because of fragmented, chaotic price structuring, and the proliferation of different classes of cigarettes.

84–1608. Tabak Journal International. **Statistics. Production of Unmanufactured Tobacco.** *Tabak Journal International* 2: 166-168, April 1984.

Figures on worldwide production of unmanufactured tobacco in metric tons (mt) are provided for 1978 through 1983. Totals are tabulated for 24 countries in Africa, 11 in South America, 14 in the North American hemisphere, 25 in Asia, and 18 in East and West Europe and for Russia, Australia, and New Zealand. The only overall decline in production was registered in North American countries (Canada, Cuba, the Dominican Republic, and the United States showed fluctuating but overall decreases and Mexico registered little change), and in Russia, where 1983 figures are slightly lower than 1978, but higher than in 1980 to 1982. All other areas reported higher figures in 1982 compared with 1978, although production fell in some individual countries (Burma, Japan, Republic of Korea, Pakistan, Turkey, France, and Australia among the larger producers). China produced 1,495,000 mt in 1983, down from 2,078,000 in 1982, but an increase over the 1,242,000 in 1978, making it the largest producer in the world.

84–1609. Tabak Journal International. **Trends Towards Stabilization on the Polish Tobacco Market.** *Tabak Journal International* 2: 93, 96-97, April 1984.

After 3 years during which supply and demand could be adjusted only by governmental measures, the Polish tobacco market appears to have returned to normal. Contrary to the fears of many, the abolition of cigarette rationing in 1983 did not lead to a further collapse of the market. However, the situation is not completely stable. Government control of cigarette sales, which lasted almost 18 months, has made a reliable assessment of market conditions almost impossible. Hoarding, by smokers anticipating the 40 to 60 percent price increase following the lifting of rationing in March 1983, created a bizarre situation; sales in the first quarter (under rationing) were higher than sales in the second quarter, leaving retailers, wholesalers, and manufacturers with surpluses. The industry needs to obtain details in 1984 to determine the true level and structure of demand. Since supplies are not expected to increase to any extent, further price adjustments may be needed. Even if there is a big increase in demand, return to rationing is not anticipated. (Auth. Abs. Mod.)

84–1610. Tabak Journal International. **Reynolds Counters Anti-Smoking Legislation.** *Tabak Journal International* 2: 102, April 1984.

R. J. Reynolds Tobacco Co. has launched a series of advertisements entitled "Can we have an open debate about smoking" that will appear in various national newspapers and news magazines and will cover subjects such as passive smoking, smoking by youth, smoking courtesy, and health issues. Designed to dispel antismoking activists' accusations, Reynolds is attempting to substitute rational discussion for the emotionalism currently surrounding smoking. Reynolds will emphasize that it does not believe young people should smoke; smokers and nonsmokers can work through mutual cooperation without legislative interference; and the causes of diseases statistically linked to smoking are an open controversy rather than a closed case. Also, during the last three decades the tobacco industry as a whole spent more than 110 million dollars for independent smoking and health research.

84–1611. Tabak Journal International. **British Leaf Buyers Have Turned to New Markets.** *Tabak Journal International* 2: 151, 153, April 1984.

Because of the intense competition between British manufacturers, buyers are trying to cut costs by finding cheaper leaf rather than buying cheaper grades in costlier traditional markets. In 1975, 41 percent of Britain's leaf came from North America, but the proportion had dropped to 21 percent by 1982. Britain now ranks fourth among U.S. buyers. Reduction in purchases is due partly to a reduction in the demand for tobacco. Tax-induced declines in cigarette consumption have cut leaf imports from all sources by 11 percent over the past 8 years. However, purchases in East Asia, Central Africa, and Latin America, especially Brazil, have increased. Also, with the end of sanctions, British buyers are returning to Zimbabwe (formerly Rhodesia), purchasing 15.5 million kg in 1980 and even more in 1981 and 1982. Britain is also buying from Zimbabwe's neighbor Malawi, now among the top 10 exporters in the world.

84–1612. Tabak Journal International. **Steady Rise in Tobacco Production and Consumption.** *Tabak Journal International* 2: 138, 140, 142-143, April 1984.

Cigarette consumption is increasing 4.8 percent annually in Yugoslavia, in spite of price increases and a media advertising ban. Among the 35 percent of adults who smoke, the average age is dropping and consumption among women is increasing. Luxury brands are preferred, and in response to changing tastes, manufacturers are reducing tar and nicotine content. Although pesticide residues and quality standards are regulated (for instance, a low-tar cigarette must not exceed 15 mg tar), labels showing tar and nicotine content are not required. Four types of tobacco are grown in Yugoslavia (oriental, semi-oriental, Burley, and Virginia). While cultivation is almost completely by private growers, all other areas of the tobacco economy belong to the State; 65 organizations are involved in purchasing and processing. Exports account for 35 to 40 percent of leaf produced. Potential production is estimated at around 150,000 tons. (Auth. Abs. Mod.)

84–1613. Thassitis, B. **Greek Cigarette Sales Gain, Largely Through Foreign Blended Brands.** *Tobacco International* 186(5): 14-17, March 2, 1984.

The strong increase in cigarette sales in 1983 (estimated at 5.1 percent) follows a 2 percent increase in 1981 and 7.8 percent in 1982, and was achieved despite long strikes at two plants. Growth is attributed to trends in the Greek market: increased smoking by women and younger age groups, reduction in weight of Greek cigarettes, and relatively low prices. Quantitative restrictions have reduced imported brands to 80 percent of the 1982 volume. Finance ministry data show total sales reached 25,336 million pieces in 1983, compared with 24,095 million in 1982, 22,356 million in 1981, and 21,902 million in 1980. Sales of nonfiltered cigarettes declined, but a steady increase was registered by filter cigarettes. There are six Greek cigarette manufacturers, and a large portion of the increase in sales is of their foreign-blended brands; however, a slowdown in consumption is expected, due to a disproportionately greater price increase for these cigarettes in January 1984.

84–1614. Thassitis, B. **Cigarette Consumption Increase Continues in Greece.** *Tabak Journal International* 2: 116, 118, April 1984.

Cigarette sales in Greece continued to increase during 1983 at a rate of 5.1 percent. Sales had been increasing since 1971 (15,301 million pieces) until a stagnation occurred and sales remained below 21,949 million pieces during the 1978 to 1980 period. In 1981, however, a 2 percent rise was seen, followed by a 7.8 percent jump in 1982. Increasing sales are generally attributed to onset of smoking among women and younger age groups, relatively low prices, and restrictions on imports. The market share of filtered and luxury-type cigarettes is increasing steadily, at the expense of plain cigarettes, which declined 8.9 percent. The market shares and sales trends of the six Greek manufacturers, and the various brand categories, are detailed.

84–1615. Tobacco International. **The Indonesian Kretek Industry, Domestic and Export.** *Tobacco International* 185(26): 59-66, December 23, 1983.

Indonesian clove cigarettes, or kretek, usually contain 60 percent tobacco and 40 percent clove and weigh an average of 1.5 g. In 1982, the three largest producers manufactured 43.1 billion kretek (72.9 percent of the country's production), a substantial increase over the 40.8 billion (66.2 percent) produced in 1981. Clove cigarettes, with production about double that of pure tobacco cigarettes, are consumed mostly by the domestic market, with the largest importers being Malaysia, Hong Kong, and the United States (mainly Hawaii, San Francisco, and New York). Consumers in the United States, where imports have doubled every year since 1977, are almost exclusively college students and young adults, and half are female. The number of clove cigarette factories in Indonesia was reported as between 226 and 283 in 1979. Although the industry is growing rapidly, the number of companies is declining due to the domination of the three manufacturers backed by capital, marketing networks, and extensive marketing campaigns.

84–1616. Tobacco International. **The Economist Intelligence Unit Tobacco Study, I. Introduction; and the Socio-Economic Impact of Leaf Tobacco.** *Tobacco International* 186(7): 10, 12, 15, 17, 19, 21-22, 24, 26, 28, 30, 34, 36, 38-39, March 30, 1984.

Studies of five agricultural areas (in Brazil, Costa Rica, India, Malaysia, and Nigeria) contrasted the importance of tobacco as a crop and wealth of the regions, climate, and lifestyles. Santa Cruz, Brazil, one of three claimants for the "World Tobacco Capital" title, employs up to 10,000 persons directly, and tobacco growing has had a widespread economic multiplier effect. On a smaller scale, tobacco-growing areas in other countries realize similar prosperity. Evidence is cited to show that tobacco growing need not conflict with existing or increased food production. International tobacco manufacturers frequently undertake development of neglected land and of export industries. Although profit is the principal motive, the community benefits, particularly in the elimination of foreign currency payments for tobacco imports.

84–1617. Tobacco International. **Generics, Store Private Labels, and Eve Made '83 a Banner Year for L and M.** *Tobacco International* 186(6): 43-46, March 16, 1984.

In 1983, for the third straight year, Liggett and Myers Tobacco Company (LM) enjoyed large increases in unit sales, due largely to the success of generic and private label cigarettes and the female-oriented brand Eve. While other major cigarette manufacturers braced for a sales slump when the Federal excise tax on cigarettes doubled in January 1983, LM pushed its generics. Sales doubled over the first 6 months, and generics claimed a 3 percent market share by year's end. A quality seal program launched in November further assured the consumer of a top quality product. The Eve brand reported a 12 percent increase in sales over 1982, and was joined by a 100s version. LM, which is trying to build its market share by looking for specific market segments rather than competing directly with giants such as R. J. Reynolds and Philip Morris, is turning to the international sphere despite having sold international trademarks in 1979. The approach is based on custom manufacturing: LM tailors the generic and private labels, works with distributors overseas to develop a blend for a particular market, and provides assistance with packaging and advertising.

84–1618. Tobacco International. **The Economist Intelligence Unit Tobacco Study, II. The Role of the International Tobacco Manufacturing Companies.** *Tobacco International* 186(8): 6-8, 10, 12-14, April 13, 1984.

The role of large manufacturing companies is explored in part II of a three-part series on tobacco's contribution to the economic and social development of Third World countries. Six important functions are identified: (1) pioneering and developing new areas for tobacco production and subsequent economic growth; (2) providing extension services and materials (usually at cost) to farmers; (3) buying the leaf produced; (4) supporting (overall) family-owned farms; (5) providing financial support and investment on a variety of levels; and (6) conducting research and training. It is concluded that newly developed tobacco growing areas are not "exploited" by the companies, although their operations are also not altruistic. However, long periods of stability are required for companies to recoup initial investments of skilled labor and capital, and genuine concern for the community is evident in the case study areas.

84–1619. Tobacco Reporter. **Canary Islands: Industry Revitalization.** *Tobacco Reporter* 111(5): 30, May 1984.

Although the Canary Island domestic tobacco industry is small, in spite of many tourists, tobacco manufacturing is one of the main sources of income, with exports going primarily to mainland Spain. However, marketing changes have reduced Spanish imports of black tobacco cigarettes from 40 percent of the market in 1972 to only 17 percent in 1982. In an effort to revitalize the Canary Island industry, the Spanish Government helped to consolidate many small manufacturers into the four remaining primary companies. Also, a new law (1984) will permit the import of blond cigarettes from island manufacturers, which will aid economic recovery. It appears likely that the Canary Islands will be given some form of special exemption status that will enable it to be part of the European Economic Community and yet retain its free port status, one of the few advantages left to island manufacturers.

84–1620. Toussaint, W. D. **The Flue-Cured Tobacco Program, and Where It May Be Going.** *Tobacco International* 186(9): 36, 38, 40, April 27, 1984.

Problems facing the flue-cured tobacco industry are discussed, noting that several factors are involved: cigarette consumption is declining, exports are decreasing and imports increasing, and the No Net Cost program is having an effect on the Flue-Cured Tobacco Stabilization Corp. that handles sales. The combination of factors suggests that stabilization may collapse and there would be no flue-cured program as it exists today. Possible changes in prices, production, quotas, land values, and geographic movement of growing areas are reviewed, noting that the effects, particularly in North Carolina, may be significant.

84–1621. Weiss, E. A. **Tobacco Production in the USSR Adapts to Changing Consumer Tastes.** *Tabak Journal International* 2: 132-133, April 1984.

See also, **84–1624**

Consumer cigarette preferences are changing in Russia, with the traditional papirosy (a cigarette with a hollow tube mouthpiece) giving way to western-type (milder blends) cigarettes. The area planted to tobacco, which increased from 160,000 ha to over 190,000 ha during the 1970s, has since remained relatively static. The crop is mostly oriental, with only a small portion of Virginia types. Imports of leaf have varied considerably; from 66 kt in 1979 to 124 kt in 1983. One forecast stated that leaf imports are to be reduced, but domestic tobacco consumption is reportedly still rising. How the demand will be met is unclear, since cigarette imports doubled in the 1970s and recently increased another 20 percent to 60 billion pieces. To help handle the demand, re-equipment of local manufacturing plants and expansion of leaf processing and flue-curing facilities is under way.

LEGISLATION

84–1622. Sumner, D. A.; Alston, J. M. **Consequences of Elimination of the Tobacco Program.** North Carolina Agricultural Research Service, North Carolina State University, Raleigh, Bulletin 469, March 1984, 59 pp.

The probable consequences of eliminating price supports and production restrictions on U.S. tobacco, as introduced to Congress in 1983, are examined. Anticipated impacts are based on (1) ownership of tobacco production rights, which is concentrated in the rural areas of the Southeast among farming and nonfarm families; (2) the probability of potential expansion of tobacco production in many of these areas; (3) demand for tobacco from exports and import replacement that would allow significant expansion given reduced prices; and (4) the low storage cost of tobacco relative to its value, which allows production in any year to be considered a source of supply in subsequent years. Given these considerations, major effects of deregulation are estimated in terms of the income and wealth of tobacco growers and allotment owners, output of tobacco and its price, farm size, and the potential for price variability and concentration of tobacco buying. Results suggest a greater possibility of industry expansion than has been previously projected; relatively few problems of price variability and relatively little effect on market power of tobacco buyers, farm size, and regional economics. However, there is the potential for serious losses to persons for whom tobacco quotas and allotments are major sources of income.

84–1623. Sumner, D. A.; Wohlgenant, M. K. **An Economic Analysis of the 1983 Increase in the Federal Excise Tax on Cigarettes.** North Carolina State University, Raleigh, Economics Research Report No. 44, April 1983, 41 pp.

Probable effects of the recent Federal cigarette tax increase are analyzed, and an equilibrium displacement model of the cigarette industry is developed. The model includes relationships for international trade in cigarettes and tobacco, Federal cigarette taxes, and market interrelationships between cigarettes and tobacco and other inputs in cigarette manufacturing. A range of values for the demand and supply elasticities is used to examine the sensitivity of the calculations. Major findings indicate that (1) cigarette prices will rise by almost the full amount of the tax increase; (2) the price increase will lead to an approximate 5 to 6 percent reduction in domestic sales, but some increase in exports will offset the decline; (3) national distribution of cigarette sales will be altered slightly because relative price increases will be greatest where current prices (and State tax rates) are lowest; (4) the effect of the tax increase on the price of domestic tobacco will be small (even holding the production of tobacco constant) because of the importance of international trade in cigarettes and tobacco, as well as substitution with other inputs; and (5) a reduction of the real support price of tobacco in response to the tax increase will cause a fall in average tobacco quota lease rates. (Auth. Abs.)

84–1624. Tabak Journal International. **China Puts Tobacco Regulations Into Effect.** *Tabak Journal International* 2: 134, 136-137, April 1984.

Regulations issued by the State Council of China cover all tobacco and cigarette production and establish the State Tobacco Monopoly Administration to administer the country's tobacco industry. The aim is to promote planned production, raise quality, improve supply, regulate consumption, and increase revenues. The administration will supervise all activities related to tobacco and cigarette production, including planning and purchase of tobacco; production and sales of cigarettes; pricing; trademark registration; transportation; production and distribution of cigarette paper, filters, and cigarette manufacturing equipment; international trade; and economic and technical cooperation with foreign countries. The provisions also stipulate a reorganization of research and technical resources to improve the quality of tobacco products and reduce the yields of tar, nicotine, and other smoke components. (Auth. Abs. Mod.)

GENERAL

84–1625. American Council on Science and Health. **Smoking and Health 1964-1984.** Proceedings of a Special Conference to Commemorate the 20th Anniversary of the First Surgeon General's Report on Smoking and Health, January 11, 1984. New York, American Council on Science and Health, 1984, 62 pp.

Statements from physicians, scientists, health educators, and private citizens participating in a special conference commemorating the 20th anniversary of the release of the first Surgeon General's Report on Smoking and Health are presented. After a short history of the antismoking campaign and perspectives on the Surgeon General's report, the following topics were addressed: (1) personal experiences with the health consequences of smoking; (2) the role of smoking in development of heart disease, cancer, and chronic lung disease; (3) adverse health effects of smoking during pregnancy; (4) children and smoking; (5) cigarette addiction; (6) antismoking public education needs; (7) interference by the tobacco industry to suppress mass media antismoking campaigns; (8) litigation against the tobacco industry; (9) health care costs stemming from development of smoking-related disease; and (10) growing recognition of nonsmokers' rights. The conference concluded by adopting a comprehensive resolution to continue a multifaceted attack against smoking.

84–1626. Bloch, H.; Pupp, R. **The Impact of Smoking on Health, Productivity, and Earnings: A New Collective Bargaining Frontier.** In: Musacchio, R. A.; Hough, D. E. (Editors). *Socioeconomic Issues of Health 1981.* Chicago, American Medical Association, 1981, pp. 35-50.

Using a 1968 data base containing only employed males between the ages of 18 and 65, it was determined that the economic cost of smoking to an average white and nonwhite American worker was 392 and 548 dollars, respectively. Estimates include the value of lost leisure time and decreased nonmarket productivity, in addition to lost labor and decreased market productivity because of smoking. A two-equation time allocation model that estimates health and wages was used. The measure of health was self-reported and calculated as the number of working hours in the year, minus 16 hours for each reported workday lost because of illness. The individual cost of smoking was defined as the difference between total income of an average smoker and nonsmoker. Findings indicate that 25 and 48 health hours/year, respectively, are lost by white and nonwhite workers because of smoking. Differences in the impact of smoking on the health and income of whites and nonwhites may be due to differences in the amount or kind of cigarettes smoked or to the interaction between smoking and other environmental/personal factors.

84–1627. Cummins, K. **The Cigarette Makers. How They Get Away With Murder...With the Press As an Accessory.** *Washington Monthly,* pp. 14-24, April 1984.

In spite of the ban on television and radio advertising, fewer smokers (down from 37 percent to 29 percent between 1980 and 1982), and lowered sales, tobacco products account for 2.5 percent of the gross national product. Abuses of the ban on portraying athletes as smokers, sponsorship of athletic and cultural events, and promotions targeted at children are widespread. The industry reassures smokers by disputing evidence of damaging health effects. These violations of the voluntary guidelines are made possible by influential congressional advocates from tobacco-growing States. Manufacturers do not oppose the price support program because small, independent farmers are effective lobbyists and divert attention from the large, diversified tobacco companies. The Federal Trade Commission has little power, and the Consumer Product Safety Commission and the Food and Drug Administration lack jurisdiction. Among suggested remedies are (1) stricter Congress-mandated package warnings and mandatory compliance with advertising guidelines, (2) extension of the advertising ban to include newspapers, magazines, and billboards, and (3) better coverage of antismoking materials by the media.

84–1628. Northup, G. W. **Smoking in Hospitals: A Paradox.** *Journal of the American Osteopathic Association* 82(4): 242/43-243/44, December 1982.

The paradox of allowing hospital patients to smoke in a hospital that is treating patients with smoking-related illnesses is discussed. While availability of cigarettes has decreased, few hospitals have had the courage to ban smoking by all patients and visitors. Doctors need to take the initiative by not smoking in patients' rooms and taking a stand against smoking. The Federal Government is also part of the paradox in attitudes about smoking, spending millions of dollars on research while subsidizing the production of tobacco. A case where a worker received compensation for a problem that resulted from working with heavy smokers is cited as an example of what can be accomplished through legal channels.

84–1629. Oster, G.; Colditz, G. A.; Kelly, N. L. **The Economic Costs of Smoking and Benefits of Quitting.** Lexington, Massachusetts, D.C. Heath and Company, 1984, 176 pp.

The economic consequences of cigarette smoking, measured by the three most important parameters (coronary heart disease (CHD), chronic obstructive pulmonary disease (COPD), and lung cancer), and the benefits of quitting are analyzed. Costs vary by the disease, sex, age group, and consumption level. The cost-recovery ratio, indicating the degree of cost recovery made possible by quitting, is always less than one, since an ex-smoker's risk of developing a smoking-related disease does not return to that of a nonsmoker for years after cessation. The ratios for light and moderate smokers are uniformly lower (but maintain the same pattern) than those for heavy smokers. Ratios are highest in young smokers for CHD, but the ratio for COPD remains fairly constant throughout life for both men and women. Cost recovery at all ages is highest in the youngest group and the heaviest smokers, and declines with increasing age and with decreasing consumption. Men under 45 who smoke >2 packs/day incur an average of >56,000 dollars in additional illness costs during their lifetimes and gain benefits of about 34,000 dollars from quitting. For women, lifetime cost is >19,000 and recovery about 12,000 dollars. Even among those over age 70, benefits of quitting are 600 to 2,500 dollars for men and 400 to 3,000 dollars for women. Young heavy smokers (men and women under age 50 who smoke >2 packs/day) incur the heaviest costs from smoking and, after quitting, recover the largest amount. However, the expected costs of smoking are sizable for all ages, regardless of sex or amount smoked, and all subgroups benefit substantially by quitting.

84–1630. Pearce, F. **Britain Exports the Smoking Holocaust.** *New Scientist* 100(1386): 642-643, December 1, 1983.

A review of Britain's Royal College of Physicians' fourth report on smoking and health notes that the report contains a warning to the Third World, stating the international tobacco industry is attempting to ensure that as many people as possible take up the habit. In Great Britain, 15 to 20 percent of mortality is smoking-related (approximately 100,000 lives and 50 million workdays each year) in spite of the prevalence of low-tar cigarettes. Today, 78 percent of men in Katmandu, 80 percent in urban Senegal, 72 percent of medical students in Lagos, and 39 percent of doctors in Bangladesh are smokers. Also, average tar/cigarette is much higher, and smoking-related mortality is rising rapidly. It is noted that three of the world's six largest tobacco companies (nongovernmental) are based in Britain. Britain has provided aid for tobacco manufacture in developing countries, but has failed to provide financial support to international smoking and health activities. Voluntary agreements with the tobacco industry are considered unsatisfactory. Passive smoking, low-yield products, and smoking-related issues among women and children are also discussed in this report, the first from the Royal College since 1977.

84–1631. Schuman, L. M. **The Samuel C. Harvey Memorial Lecture: Progress and Responsibilities of Educators in Smoking Control.** *Medical and Pediatric Oncology* 11(6): 375-382, 1983.

The problem of tobacco use is not only one of excess mortality but also one of premature mortality, particularly among men between the ages of 35 and 59. It has been calculated that an excess of 33 percent of all deaths among those aged 35 to 59 and 44 percent among those 45 to 49 years of age are smoking-related. Besides early mortality, an estimated 11 million extra cases of chronic illness occur annually because of cigarette smoking. Since the 1964 Surgeon General's report, which served to significantly weaken the tobacco industry subsystem and enhance the health forces, a decline in smoking has been observed through 1982, to the lowest level since 1950. Beyond educating the public, the Surgeon General's reports have spurred legislation, ranging from labeling of cigarette yields to mandating health education campaigns. It is emphasized, however, that the concept of primary prevention through smoking cessation, and its role in medical school curricula, is still sadly lacking. The potential impact of practitioners is stressed, noting that treatment of a patient's smoking habit, regardless of his or her complaint, would convert medical care to comprehensive health care. Tobacco industry opposition, consumption trends in different age, sex, and ethnic groups, and antismoking legislation are discussed. Also, a Roper Organization report on public attitudes toward smoking, sponsored by the tobacco industry, is summarized.

84–1632. Sigmon, T. **Cigarette Smoking Injuries: A Theory for Recovery Against the Federal Government.** *Trial* 19(4): 64-67, 96-97, April 1983.

The Federal Government has assumed irreconcilable roles in the tobacco controversy, advising against smoking but providing free services to tobacco farmers through the Department of Agriculture. The Government has made a stronger financial and philosophical commitment to the perpetuation of the tobacco habit than to its extinguishment. However, by entering the market as a major buyer and seller of tobacco, the Government may be legally liable for injuries suffered by consumers. A number of traditional product liability theories by which the Government might be found liable are discussed, including negligence claims arising out of its involvement with the tobacco industry, as an imputed cigarette manufacturer or endorser of certain brands (FTC name and test results displayed on packages), and failure to adequately warn consumers of the dangers of the product. The Government undoubtedly would assert the defenses of the assumption of risk and contributory negligence in an action by an injured tobacco consumer, but such assertions could be contested.

84–1633. Whelan, E. M. **Big Business vs. Public Health: The Cigarette Dilemma.** *USA Today* 112(2468): 61-66, May 1984.

Despite current efforts to prevent environmentally induced disease, some 400,000 Americans will die in 1984 from diseases directly associated with cigarette smoke. Apathy toward cigarettes as a major public health hazard stems from (1) association of cigarette smoking with pleasure, prestige, relaxation, and the carefree American lifestyle; (2) the physically addictive aspects of smoking; (3) psychological defense mechanisms used by smokers to avoid coping with known harmful effects; (4) reluctance of nonsmokers to pressure smokers to quit; and (5) unprecedented economic power exerted by the tobacco industry. To combat the complex cigarette smoking phenomenon, it must be recognized as unique, meriting unique solutions, including the following: (1) smokers and companies that market cigarettes, rather than the entire populace, should assume the economic responsibilities associated with the habit; (2) both voluntary and government agencies should escalate attempts to publicize warnings on the dangers of smoking; (3) cigarette advertisements should be toned down or eliminated; and (4) a smoking substitute should be developed that, when used with behavior modification, would have a significant success rate in breaking the addiction cycle.

See also **84–1622**

AGRICULTURE

84–1634. Court, W. A.; Elliot, J. M.; Hendel, J. G. **Influence of Applied Nitrogen Fertilization on Certain Lipids, Terpenes, and Other Characteristics of Flue-Cured Tobacco.** *Tobacco International* 186(13): 91-94, June 8, 1984.

A field experiment was conducted in 1980 and 1981 on Fox loamy sand in Ontario to study the effects of different rates (16.8, 33.6, and 50.4 kg/ha) of nitrogen (N) fertilization on individual constituents of flue-cured tobacco (*Nicotiana tabacum* L., cv Nordel). Nitrogen fertilization increased yield, total alkaloids, hexane extracts, cytoplasmic lipids, individual pigments, neophytadiene, and solanesol. Increasing the rate of N fertilization decreased grade index, reducing sugars, and duvatrienediols. Nitrogen fertilization had no influence on maturity index, surface waxes, hydrocarbons, and total phytosterols. (Auth. Abs.)

CHEMISTRY

84–1635. Adams, J. D.; Lee, S. J.; Hoffmann, D. **Carcinogenic Agents in Cigarette Smoke and the Influence of Nitrate on Their Formation.** *Carcinogenesis* 5(2): 221-223, February 1984.

The nitrate level of nonfilter reference cigarettes was elevated from 0.52 percent to 1.2, 1.8, 2.4, and 3.05 percent, respectively, by addition of sodium nitrate. Data from mainstream smoke analyses showed that yields of carbon monoxide and carbon dioxide were not significantly altered by nitrate elevation. Tar, nicotine, benzo(*a*)pyrene (BaP), and catechol in mainstream smoke were reduced, while yields of nitrogen oxides (NO_x), volatile N-nitrosamines (VNA), and tobacco-specific N-nitrosamines (TSNA) were significantly increased. On the basis of previous bioassays with smoke condensates from high-nitrate cigarettes, it was expected that the cutaneous tumorigenicity of these tars would be reduced because of lower levels of BaP (and other carcinogenic polycyclic aromatic hydrocarbons) and catechol. However, the total carcinogenic potential of whole smoke from high-nitrate cigarettes is considered to be significantly increased owing to elevated yields of NO_x, VNA, and TSNA. Nitrosamines are regarded as a major group of carcinogens in tobacco smoke, and nitrogen oxides are the most important precursors for the endogenous formation of N-nitrosamines upon smoke inhalation. Findings support the recommendation that the nitrate content of tobacco products should be reduced. (Auth. Abs.)

84–1636. Arrendale, R. F.; Chapman, G. W.; Chortyk, O. T. **Gas Chromatographic Analyses of Fatty Acids on Laboratory-Prepared Fused Silica Silar 10C Capillary Columns.** *Journal of Agricultural and Food Chemistry* 31(6): 1334-1338, November-December 1983.

An improved chromatographic column for analyzing fatty acids in cigarette smoke is described. Tobacco leaf contains long-chain fatty acids (C_{12}-C_{34}), 5 to 15 percent of which are transferred to cigarette smoke during smoking and contribute to the flavor and aroma of the cigarette. Plant-derived, long-chain fatty acids were readily analyzed as fatty acid methyl esters by glass capillary gas chromatography (GC-2) on columns coated with Silar 10C, a chemically inert, highly flexible fused silica capillary tubing. Laboratory methodology for the preparation of fused silica GC-2 columns was adapted to the preparation of Silar 10C capillary columns. The applicability of the columns for GC-2 analyses of fatty acids from tobacco smoke, cooking oils, and forage grasses is demonstrated, and laboratory preparation and analytical utility are described. (Auth. Abs. Mod.)

84–1637. Cabo, J.; Bravo, L.; Fernandez, F.; Jimenez, J.; Navarro, C. **Estudios Sobre Tabacos de Elaboracion Nacional. V. Determinacion Cuantitativa de los Productos de Pirolisis. [Studies on Nationally Made Tobaccos. V. Quantitative Determination of the Products of Pyrolysis.]** *Ars Pharmaceutica* 23(4): 493-499, 1982, Spanish.

Using a simple smoking machine, products of pyrolysis of 15 popular brands of Spanish cigarettes were analyzed quantitatively. Determinations were made (on 50 mm of each cigarette) of the weights of the products with and without filters (12 of 15 were filtered) and the percent efficacy of the filter calculated. Without filters, products were between 1.42 and 10.58 percent of total cigarette weight; 40 percent were <6 percent by weight. With filters, products were 0.71 to 4.76 percent by weight. Filter efficacy ranged from 20 to 63.64 percent. Alkaloids expressed as nicotine were < 10 percent in 10 brands, and from 13.6 to 30 percent in the other 5. Determinations were highly reproducible.

84–1638. Frecker, R. C.; Pischkitl, H. **Constituents of Cigarettes From Developing Countries: Nicotine, Tar, and Carbon Monoxide Values for 50 Brands Selected by the World Health Organization.** World Health Organization, Document No. WHO/SMO/84.4, 1984, 12 pp.

Data from 50 brands, which were conditioned, machine-smoked, and analyzed for nicotine, dry tar, and carbon monoxide, show that, in general, the cigarettes are very similar to those available in North America, Great Britain, and Europe. The majority were filtered (the only kind analyzed in this report), and mean length was 83.45 mm. Mean length of the tobacco column was 64.69 mm; average filter length was 18.76 mm. There were no dramatic differences found in a comparison of 16 brands with 36 similar U.S. brands. Observations on individual brands note that (1) the Legales SN from Argentina contained no nicotine, but had a relatively high tar yield/cigarette (22.1 mg); (2) Marven Gold from Pakistan had the highest nicotine level (2.4 mg/cigarette); (3) Vila Rica from Brazil had the second highest nicotine yield/cigarette, but the highest on a per-puff basis; and (4) a number of brands from several countries were rated mild. Yields for most of the 50 brands were well within those published by the U.S. Federal Trade Commission. One brand from Pakistan and one from Brazil were the only exceptions.

84–1639. Gutenmann, W. H.; Lisk, D. J.; Hoffman, D.; Adams, J. D. **Selenium in Particulates and Gaseous Fractions of Smoke From Cigarettes Prepared From Tobacco Grown on Fly-Ash-Amended Soil.** *Journal of Toxicology and Environmental Health* 12(2/3): 385-393, August-September 1983.

Cigarettes prepared from tobacco grown in pots of soils containing 20 percent soft coal fly ash were smoked and the mainstream and gaseous fractions were analyzed for total selenium concentration. Fly-ash-grown and control (soil-grown) tobaccos contained, respectively, 0.79 and 0.03 ppm selenium. Quantities of selenium (ng/cigarette) found in the mainstream particulate and gaseous fractions were, respectively, 62.4 and 246.0 for fly-ash-grown and 8.6 and 12.0 for control treatments. Relatively little information is available on the toxicity of selenium compounds through inhalation, but selenium compounds at concentrations present in urban atmospheres probably do not contribute significantly to human organ burdens. While high intake can be lethal, selenium's protective effects against toxic elements such as mercury and cadmium have been demonstrated. On the basis of a normal dietary intake of selenium, the additional exposure from cigarette smoking would not appear to contribute a hazard. Studies of the absorption, retention, effects, metabolism, and excretion of selenium in the body are reviewed. (Auth. Abs. Mod.)

84–1640. Masuo, Y.; Kaneda, T. **Tobacco no Shizennenshyo chu no Juryonenshyo Sokudo to Sennen Sokudo no Kankei. [Relation Between the Mass and the Linear Rate of Burn During the Smouldering of Cigarette.]** *Scientific Papers of the Central Research Institute, Japan Tobacco and Salt Public Corporation* 125: 29-32, 1983, Japanese.

Experimental data confirmed that mass rate of static burn was independent of cigarette weight and represented the loss of combustible cigarette mass. Linear rate of static burn was expressed in an equation that related linear burning rate to mass burning rate, cigarette length, cigarette weight, and residue ratio under static burn conditions. Good agreement was obtained between measured and predicted linear rate of static burn for flue-cured, Burley, and incense stick cigarettes. It is concluded that linear burn rate can be calculated from mass rate regardless of the weight of the cigarette. (Auth. Abs. Mod.)

84–1641. Matsukura, M.; Takahashi, K.; Ishiguro, S.; Matsushita, H.; Miyauchi, N. **Composition of Semivolatiles From Roasted Tobacco.** *Agricultural and Biological Chemistry* 47(10): 2281-2285, 1983.

A preliminary study on the composition of the semivolatiles included in the volatiles from roasted tobacco was conducted to examine the applicability of the exhaust gas components from the tobacco-roasting process as cigarette flavor ingredients. A relatively large quantity of sugar pyrolysates (furans, volatile ketones, and lactones) in addition to lower fatty acids and the components of essential oils were found in the condensate of the volatiles from roasted flue-cured tobacco. Condensate from roasted Burley tobacco contained nicotine and neophytadiene as major components, and a number of other components of essential oils were also found. The compounds in the condensate from flue-cured tobacco were assumed to contribute to its burnt-sugar-like aroma. A sweet note of the condensate from Burley tobacco might be attributable to the high concentration of the components of essential oils. (Auth. Abs.)

84–1642. Osterdahl, B.-G.; Slorach, S. A. **Volatile *N*-Nitrosamines in Snuff and Chewing Tobacco on the Swedish Market.** *Food and Chemical Toxicology* 21(6): 759-762, December 1983.

Snuff and chewing tobacco on the Swedish market during 1981 and 1982 were analyzed for volatile N-nitrosamines using gas chromatography-thermal energy analysis. Detectable levels of N-nitrosodimethylamine and N-nitrosopyrrolidine were found in nearly all of the 36 samples analyzed. N-nitrosopiperidine and N-nitrosomorpholine were also found in some samples. The total concentration of volatile N-nitrosamines found in any sample was between 0.5 and 145.9 μg/kg wet weight. Storage of snuff in unopened boxes for 20 weeks at -20, +2, or +23°C resulted in small but statistically insignificant changes in volatile N-nitrosamine content. Storage for 20 weeks at +2°C in boxes that had been opened and resealed led to a larger but statistically insignificant increase in mean level of total volatile N-nitrosamines. Levels of volatile N-nitrosamines in snuff were significantly ($p < 0.05$) lower than those found in 1979. It is estimated that in Sweden today the use of snuff can increase the normal intake of N-nitrosamines by about 10 to 100 percent. (Auth. Abs. Mod.)

84–1643. Park, J. W. **Analyses of Phenolics in Cigarette Smoke by GC-MS With the Multiple Ion Selection Technique.** *Archives of Pharmacal Research* 5(2): 71-77, December 1982.

Improvements in the analytical methodology used in the gas chromatographic/mass spectral (GC/MS) analysis of phenolic compounds from cigarette smoke are described. For the direct analysis of crude samples, pyridine extraction and the glass capillary column GC were used for the separation of phenolics as trimethylsilyl derivatives. The separations of cigarette smoke on Carbowax 20M and SE-54 wall-coated open tubular columns are given. Improved methodology for routine quantitation of identified components using the computer-controlled multiple ion selection technique of MS is presented. Considerations pertaining to routine analyses of a multitude of complex smoke samples are also discussed. (Auth. Abs.)

84–1644. Seeman, J. I.; Secor, H. V.; Howe, C. R.; Chavdarian, C. G.; Morgan, L. W. **Organometallic Methylation of Nicotine and Nicotine *N*-Oxide. Reaction Pathways and Racemization Mechanisms.** *Journal of Organic Chemistry* 48(25): 4899-4904, December 16, 1983.

The reaction of nicotine with methyllithium leads to 2-methylnicotine as a major product in addition to the 4- and 6-methylnicotines. Reaction of nicotine N-oxide with methylmagnesium bromide furnishes both 2- and 6-methylnicotine. The product composition of these reactions is strongly dependent on experimental conditions, and the effects of solvent, temperature, and relative reagent concentration are presented. Methyllithium reactions lead to partially racemized methylnicotines, and the recovered nicotine is often nearly optically pure. Independently, (*S*)-(-)-6-methylnicotine was treated with methyllithium and recovered with complete retention of optical activity. Results suggest that the loss of optical purity in the formation of methylnicotines in the methyllithium reactions occurs during the reaction itself and is not due either to racemization of the starting material or to subsequent racemization of the initially formed product. (Auth. Abs.)

PHARMACOLOGY AND TOXICOLOGY

84–1645. Bartelik, S.; Ziolo, H.; Bartelik, M. **Fizjologiczne i Biochemiczne Parametry Krwi Osob Niepalacych i Palacych Papierosy. [Physiological and Biochemical Blood Parameters in No-smokers and Tobacco Smokers.]** *Polski Tygodnik Lekarski* 39(1): 7-8, January 1984, Polish.

Comparisons of hematologic parameters of nonsmokers and smokers showed significantly higher mean levels of immunoglobulin (Ig) A (252.6 in nonsmokers vs. 172.0 mg/dl in smokers), IgM (185.3 vs. 131.6 mg/dl), IgG (1,319.7 vs. 1,066.9 mg/dl), total lipids (6.27 vs. 5.74 g/l), and β-lipoproteins (3.98 vs. 3.42 g/l) among nonsmokers. Hematocrit (46.5 vs. 48 percent), erythrocyte count (5.3 vs. 6.5 million/mm^3), and α-2 macroglobulin (197.5 vs. 217.1 mg/dl) were significantly higher in smokers. (Auth. Abs. Mod.)

84–1646. Bend, J. R.; Foureman, G. L.; Ben-Zvi, Z.; Albro, P. W. **Heterogeneity of Hepatic Aryl Hydrocarbon Hydroxylase Activity in Feral Winter Flounder: Relevance to Carcinogenicity Testing.** In: Hoover, K. L. (Editor). *Use of Small Fish Species in Carcinogenicity Testing.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, pp. 359-370.

Hepatic microsomes from winter flounder, treated with polycyclic aromatic hydrocarbon inducers, had elevated activities of benzo(*a*)pyrene hydroxylase (AHH) and 7-ethoxyresorufin deethylase (7-ERD). When electrophoresed, they showed a novel or enriched polypeptide species with a monomeric molecular weight of approximately 57,000. Results are consistent with inductive responses already characterized in several mammalian and fish species. However, when the urinary clearance of 4-chlorobiphenyl was studied in untreated flounder, wide variations (up to 20-fold) among fish were noted. Based on the response of AHH activity to 7,8-benzoflavone (ANF) added in vitro, flounder could be divided into two groups; one had high hepatic AHH activity inhibited by ANF, the other had low AHH activity enhanced by ANF. Examination of winter flounder (>400 total) over four experimental seasons demonstrated this variability of hepatic AHH activity to be a recurrent characteristic of the flounder population in waters around Mount Desert Island (Maine). Hepatic AHH activities did not correlate well with any physical parameter (e.g., liver, gonad or body weight, length, or sex) or the cytochrome P-450 content of hepatic microsomes. The variation in response appears to be related to individual exposure level (dose) or sensitivity, or both. The possibility of heterogeneity of hepatic monooxygenase activities in teleost species should be considered in development of experimental models for carcinogenesis testing in fish. (Auth. Abs. Mod.)

84–1647. Bernard, A.; Lauwerys, R. **Cadmium in Human Population.** *Experientia* 40(2): 143-152, February 15, 1984.

Tobacco smoke as a route of exposure is included in a summary of current knowledge of the metabolism and toxicity of cadmium (Cd) in man. Cigarettes generally contain 1 to 2 μg Cd, of which 10 percent is absorbed by the lung. Thus smokers at age 50 or 60 have an average Cd body burden twice as high as nonsmokers. One study estimated an average level at age 50 of 35.5 mg in smokers compared with 19.3 for comparable nonsmokers. Smoking at a workplace contaminated with Cd also increases Cd intake. The metabolism, absorption (inhalation, ingestion, and body distribution), and excretion of Cd are discussed, as are problems in evaluating exposure. The acute and chronic effects of Cd exposure are considered, including effects on the kidneys, lungs, and bones. The role of Cd in hypertension and possible carcinogenic effects are also reviewed.

84–1648. Bond, J. A.; Mitchell, C. E.; Li, A. P. **Metabolism and Macromolecular Covalent Binding of Benzo(*a*)pyrene in Cultured Fischer-344 Rat Lung Type II Epithelial Cells.** *Biochemical Pharmacology* 32(24): 3771-3776, 1983.

Cultures of a recently developed rat epithelial cell line, with morphological characteristics suggestive of type II cell origin (LEC cells), were incubated for 24 hours with 5 μM (^{14}C)benzo(*a*)pyrene (BaP) and analyzed for organic and water-soluble metabolites. LEC cells metabolized BaP to BaP-7,8-diol and BaP-9,10-diol with total rates of formation measured at 500 to 600 pmoles/10^6 cells/24 hours. BaP-9,10-diol was the major metabolite, accounting for about 80 percent of the BaP metabolized. Enzyme hydrolysis studies revealed the presence of small quantities (<20 percent of BaP metabolized) of the glucuronide conjugates of BaP-7,8-diol and 9-hydroxy-BaP. Pretreatment of LEC cells with coal gas condensate, benz(*a*)anthracene, or diesel exhaust particle extract (DEP) prior to incubation with BaP resulted in a twofold to fivefold increase in overall rates of BaP metabolism. The largest increase in covalent binding of (^{14}C)BaP equivalents to LEC macromolecules was seen after LEC cells were pretreated with DEP (threefold). Data suggest that lung epithelial cells may play an important role in the biological fate of inhaled xenobiotics. (Auth. Abs. Mod.)

84–1649. Brazell, R. S.; Stiff, A. C.; Henderson, G. M.; Jenkins, R. A.; Romig, P. L.; Auerbach, O. **Plasma Nicotine and Cotinine in Tobacco Smoke Exposed Beagle Dogs.** *Toxicology and Applied Pharmacology* 73(1): 152-158, March 30, 1984.

Results indicate that nicotine may be useful as a relative index of smoke exposure because average blood concentrations were related to number of cigarettes and level of nicotine delivery. Nicotine and cotinine were determined in plasma samples from 87 beagle dogs chronically exposed to smoke from cigarettes with three different levels of nicotine. Controls were 18 sham-exposed animals. Smoke, administered through permanent tracheostomas via cuffed tracheostomy tubes, was generated from reference cigarettes under standard puffing parameters by ADL-II smoking machines. Dogs were exposed for an average of 2 years prior to sample collection. Nicotine concentrations prior to daily exposure were, in ng/ml, 10±8 in the high-level group, 8±4 in the medium-level group, 8±7 in the low-level group, and 7±3 in controls. After three cigarettes, values were 120±60, 46±25, and 17±13, respectively; after 12 cigarettes, levels were 162±42, 74±42, and 20±10, respectively. Variation was high (both animal-to-animal and day-to-day in the same animal). Cotinine increased more slowly than nicotine and metabolized more rapidly than in humans. (Auth. Abs. Mod.)

84–1650. Bukowskyj, M.; Nakatsu, K.; Munt, P. W. **Theophylline Reassessed.** *Annals of Internal Medicine* 101(1): 63-73, July 1984.

The influence of smoking is included in a review of theophylline therapy research over the past 5 years. A variety of smoke components (nicotine, benzpyrenes, 3-methylcholanthrene) induce high rates of theophylline clearance. Smokers' clearance rates are reported at 60 to 100 ml/kg/hr, compared with rates for nonsmokers of 30 to 50 ml/kg/hr. A correlation has been found between serum thiocyanates and theophylline clearance, suggesting a dose—response relationship. Duration of the enzyme-induction effect after cessation of smoking is unclear. The influence of cigarette smoke often overrides other factors that reduce clearance, such as age, congestive heart failure, or erythromycin (but not oral contraceptive use or hepatic cirrhosis). Smoking does not appear to affect theophylline pharmacokinetics. Other factors influencing theophylline metabolism are reviewed, and theophylline's mechanism of action, assays, interactions with other drugs, effects in pregnancy, overdose, adverse effects, and role in current bronchodilator therapy are described.

84–1651. Cena, V.; Garcia, A. G.; Montiel, C.; Sanchez-Garcia, P. **Uptake of (^{3}H)-Nicotine and (^{3}H)-Noradrenaline by Cultured Chromaffin Cells.** *British Journal of Pharmacology* 81(1): 119-123, January 1984.

Three-day-old cultured bovine adrenal chromaffin cells, incubated at room temperature with Krebs-HEPES solution containing different concentrations of (^{3}H)-nicotine, took up and retained increasing amounts of the drug by a mechanism that did not saturate. Concentrations of cold nicotine as high as 100 μM failed to alter the amount of (^{3}H)-nicotine retained by cells. Imipramine, cocaine, tetracaine, and mecamylamine, at concentrations (10 μM) that blocked the catecholamine secretory effects of nicotine completely, also did not modify the uptake of (^{3}H)-nicotine. However, both imipramine and cocaine drastically inhibited (^{3}H)-noradrenaline uptake by cells in a concentration-dependent manner (IC_{50}s of 0.08 and 1 μM, respectively). Data indicate that the secretory effects of nicotine are not coupled to its previous uptake into cells and suggest a site of action for nicotine located in or at the surface of the chromaffin cell membrane. (Auth. Abs.)

84–1652. Chan, S. M. **Radioiodinated Nicotine and Nicotine Analogs: Potential Cerebral Functional Imaging Agents.** Doctoral Dissertation, University of Oklahoma, Norman, University Microfilms International 84-07275, 1984, 169 pp.

Radiolabeled (iodine) nicotine and nicotine analogs were synthesized, and biodistribution and uptake in rat brain were studied and compared with other pharmaceuticals. All of the compounds synthesized (^{125}I-5-iodonicotine, ^{125}I-3-(methylpropylaminomethyl)-5-iodo-pyridine, ^{125}I-3-(diethylaminomethyl)-5-iodo-pyridine, ^{125}I-3-(isopropylaminomethyl)-5-iodo-pyridine, and ^{125}I-3-(diisopropylaminomethyl)-5-iodo-pyridine) exhibited log P_{oct} (partition coefficient for octanol/buffer) values ranging from 0.92 to 2.16, well within the reported range for brain uptake of radiopharmaceuticals. Biodistribution studies in rats showed that all crossed the blood--brain barrier, with a blood:brain ratio of 6.0 at 2 minutes after administration. However, retention time in the brain was short, probably because the bulky iodine atom prevented binding of the base nicotine molecule to cerebral receptors. Uptake and washout in the adrenal gland were similar to that of the brain, but were not influenced by protein binding.

84–1653. Crystal, G. J.; Downey, H. F.; Adkins, T. P.; Bashour, F. A. **Regional Blood Flow in Canine Brain During Nicotine Infusion: Effect of Autonomic Blocking Drugs.** *Stroke* 14(6): 941-947, November-December 1983.

Radioactive microspheres (15 μ) were used to measure regional cerebral blood flow during intravenous infusion of nicotine (35 μg/kg/min) in anesthetized, open-chest dogs. Experiments were conducted with uncontrolled mean aortic pressure and intact autonomic receptors (series I; $n=9$) and in four groups of dogs with mean aortic pressure held constant (series II): (1) with intact autonomic receptors ($n=6$); (2) after beta adrenergic blockade ($n=8$); (3) after alpha and beta adrenergic blockade ($n=6$); and (4) after alpha and beta adrenergic and cholinergic blockade ($n=4$). In series I, nicotine raised mean aortic pressure (+72 percent) and increased flow in cerebral cortex (+67 percent), cerebellum (+38 percent), pons (+46 percent), medulla (+39 percent), and spinal cord (+48 percent). In all regions but cortex, increases in vascular resistance limited nicotine-induced increases in flow. In series II, nicotine changed flow only in cortex. Without blockade, nicotine increased cortical flow (+38 percent), but beta blockade abolished the increase. After alpha and beta blockade, nicotine again raised cortical flow (+29 percent), and additional cholinergic blockade had no effect on this response. It is concluded that nicotine causes predominant beta receptor-mediated vasodilation in cerebral cortex, but also activates alpha (vasoconstrictor) receptors and a nonadrenergic, noncholinergic vasodilator mechanism in this brain region. (Auth. Abs.)

84–1654. Dahlqvist, R.; Bertilsson, L.; Birkett, D. J.; Eichelbaum, M.; Sawe, J.; Sjoqvist, F. **Theophylline Metabolism in Relation to Antipyrine, Debrisoquine, and Sparteine Metabolism.** *Clinical Pharmacology and Therapeutics* 35(6): 815-821, June 1984.

Theophylline plasma clearance (Cl_p) and clearance of its metabolites (Cl_m) and antipyrine saliva clearance (Cl_{sal}) and clearance of its metabolites (Cl_m) were compared in a crossover study in 25 healthy subjects. Selection was made with regard to smoking status (9 smokers, 16 nonsmokers) and oxidation phenotype of debrisoquine and sparteine (6 poor metabolizers (PMs) and 19 extensive metabolizers (EMs)). Cl_p of theophylline (1,3-dimethyluric acid, 1-methyluric acid, and 3-methylxanthine) correlated ($r \geq 0.92$) to each other and to total theophylline Cl_p ($r \geq 0.97$). Smokers had higher Cl_p to all metabolites, particularly by the N-demethylation pathways. The ratio of theophylline Cl_p to antipyrine Cl_{sal} among smokers was 1.35 ± 0.38, significantly higher than in nonsmokers (0.99 ± 0.27), indicating that smoking induced theophylline metabolism more than antipyrine metabolism. After correction for the effect of smoking, there was no difference between EMs and PMs with regard to theophylline Cl_p or Cl_m. Antipyrine clearances by EMs and PMs (Cl_{sal} and Cl_m of 4-OH-antipyrine, 3-OH-methylantipyrine, or norantipyrine) also did not differ. Antipyrine Cl_{sal} and Cl_m correlated to theophylline Cl_p (r between 0.50 and 0.69). It is concluded that theophylline metabolism (N-demethylations and C-oxidation) is not under the same genetic control as sparteine and debrisoquine oxidations and that there may be a partial overlap in factors that regulate the metabolism of theophylline and antipyrine. (Auth. Abs. Mod.)

84–1655. Eldon, M. A. **The Effect of Withdrawal From Cigarette Smoking on the Pharmacokinetics of Theophylline and Pentazocine.** Doctoral Dissertation, University of Cincinnati, Ohio, University Microfilms International 83-23207, 1984, 318 pp.

Data show no significant effect of smoking withdrawal on hepatic blood flow or on the pharmacokinetics of drugs having either very low or very high hepatic extraction, suggesting that changes in dosage regimens for most drugs would not be necessary upon acute smoking withdrawal. The effects of acute withdrawal from cigarette smoking on theophylline and pentazocine pharmacokinetics were studied in healthy, young, male volunteers, using two separate crossover design clinical trials, 12 subjects each. Indocyanine green and antipyrine were administered i.v. during both trials to estimate hepatic blood flow and drug-metabolizing capacity, respectively, in withdrawn and smoking subjects. Theophylline was administered during the first trial; pentazocine during the second. Blood samples were collected following the administration of each drug from 24 hours until 36 hours after withdrawal. For all parameters, deviation of the median was <12 percent of controls, indicating that short-term smoking withdrawal had no significant effect on hepatic blood flow or hepatic drug-metabolizing activity. The finding of no changes in indocyanine green initial clearance and antipyrine clearance was predictive for no changes in pentazocine and theophylline clearances, respectively. Theophylline clearance was highly correlated with antipyrine clearance in withdrawn and smoking subjects. Hepatic blood flow rates were normal compared with values published for larger sample populations under both conditions. (Auth. Abs. Mod.)

84–1656. Elinder, C. G.; Friberg, L.; Lind, B.; Jawaid, M. **Lead and Cadmium Levels in Blood Samples From the General**

Population of Sweden. *Environmental Research* 30(1): 233-253, February 1983.

Blood lead (Pb) concentrations were significantly influenced by cigarette smoking, alcohol consumption, and sex in blood samples from 473 nonoccupationally exposed Swedish adults in 1980. Current male smokers had a median Pb level of 92 µg compared with 77 µg for nonsmokers. Corresponding values for females were 69 µg/l and 57 µg/l for current smokers and nonsmokers, respectively. Highly significant correlations were found between stated alcohol consumption and Pb in most sex and smoking categories. In Stockholm, people living in apartments close to streets with heavy traffic had slightly, but not significantly, higher Pb levels compared with people living in areas with low traffic density. Blood cadmium (Cd) levels were very strongly affected by smoking habits. A significant correlation existed between number of cigarettes consumed/day and blood Cd concentration. Median blood Cd level for nonsmoking males was 0.2 µg/l (≤ 0.2, detection limit) and for females, 0.3 µg/l. About 90 percent of all nonsmokers had Cd blood concentrations below 0.6 µg/l, whereas about 90 percent of current male and female smokers had Cd blood concentrations of 0.6 µg/l or more. (Auth. Abs. Mod.)

84–1657. Enarson, D. A.; Ferreira, P.; Hammond, M.; Chan-Yeung, M. **Routine Urinalysis in a Male Working Population: Effects of Some Personal and Lifestyle Factors.** *Clinical and Investigative Medicine* 7(1): 7-11, 1984.

Of 1,810 white male pulp and paper mill workers who returned urine samples, 398 had never smoked, 636 were ex-smokers, and 776 were smokers. The most frequent positive findings were leukocytes (78.7 percent), erythrocytes (37.3 percent), and protein (25.4 percent). Erythrocytes were significantly more common among smokers, as was proteinuria. Leukocytes were associated with age and were more common in urine of heavy drinkers. There was a significant correlation among the variables: cells were associated with protein, protein was associated with casts, and erythrocytes were associated with granular casts and oxalate crystals. Protein and granular casts were significantly associated with serum creatinine. The prevalence of blood cells varied with age, showing a bimodal distribution. Oxalate crystals were significantly more common in subjects with a history of urinary tract infection, while triple phosphate crystals were associated with a history of urinary calculi. (Auth. Abs. Mod.)

84–1658. Folsom, A. R.; Pechacek, T. F.; De Gaudemaris, R.; Luepker, R. V.; Jacobs, D. R., Jr.; Gillum, R. F. **Consumption of "Low-Yield" Cigarettes: Its Frequency and Relationship to Serum Thiocyanate.** *American Journal of Public Health* 74(6): 564-568, June 1984.

To determine the use and possible health risks of low-yield cigarettes, cigarette brands and serum thiocyanate (SCN) levels of 2,561 adult smokers (age 25-74) were investigated in population-based samples of seven upper Midwestern communities during 1980 to 1982. Brands were coded according to December 1981 Federal Trade Commission (FTC) ratings for "tar," nicotine, and carbon monoxide (CO). Compared with 1980 data from the National Center for Health Statistics for the United States as a whole, a greater proportion of smokers in these communities smoked low-yield brands. More people with higher education and more women than men smoked low-yield cigarettes. More older people (65 to 75 years) than younger people (<65 years) smoked cigarettes in the highest and lowest brand yield categories. SCN levels, adjusted for number of cigarettes smoked and for sex, was only weakly associated with brand ratings for tar ($r = 0.12$), nicotine ($r = 0.11$), and CO ($r = 0.15$). Furthermore, the gradient in SCN between lowest and highest quintiles of brand strength was <16 percent, much lower than the 300 to 500 percent gradient in smoke components implied by FTC ratings, suggesting that smoking low-yield cigarettes may not be significantly less hazardous than smoking high-yield brands. (Auth. Abs. Mod.)

84–1659. Freitas, J. **Tobacco Influence on Carboxyhemoglobin, Oxihemoglobin Dissociation and Erythrocyte Filtration.** *Acta Medica Portuguesa* Supplement 4: 73-75, April 1983.

To determine the acute effects of tobacco intoxication on blood filtration and on oxygen transport by hemoglobin, blood samples were collected from 10 subjects (5 males, 5 females) after 8 hours of abstinence from smoking (baseline), and after a 4-hour period during which each subject consumed five cigarettes. Compared with baseline values, carboxyhemoglobin concentrations increased ($p<0.001$), blood filtration rates increased ($p<0.001$), and 50 percent oxygen saturation of hemoglobin decreased ($p<0.01$) in each subject following consumption of five cigarettes.

84–1660. Gennarelli, E.; Paone, G.; Canonico, R. **Antithrombin III Determination in Smokers.** *Rassegna Internazionale di Clinica e Terapia* 63(21): 1488-1490, 1983.

The activity and blood levels of antithrombin III were determined in 20 nonsmokers, 10 smokers of 1 to 5 cigarettes/day, 10 smokers of 5 to 15 cigarettes/day, and 5 smokers consuming 15 to 25 cigarettes/day. Both activity and concentrations of antithrombin III were reduced in smokers in a dose—response manner. Antithrombin III in nonsmokers showed a mean activity of 94.5 percent and a mean concentration of 102 percent. Mean activity levels were 88, 72, and 65 percent, respectively, among smokers of 1 to 5, 5 to 15, and 15 to 25 cigarettes/day; mean concentrations of antithrombin III were 75, 65, and 60 percent, respectively. It is concluded that smoking is a risk factor for thrombosis.

84–1661. Gray, P.T.A.; Rang, H. P. **Analysis of Current Noise Evoked by Nicotinic Agonists in Rat Submandibular Ganglion Neurones.** *British Journal of Pharmacology* 80(2): 235-240, October 1983.

The kinetics of the responses of rat submandibular ganglion neurons to nicotinic agonists were investigated by analysis of agonist-induced membrane current noise. Two kinetic components are found in responses of these cells to acetylcholine, carbachol (CCh), suberyldicholine, dimethyl-4-phenylpiperazinium, and nicotine, in agreement with previous findings of two components in evoked synaptic currents in these cells. To test whether appreciable channel block occurred with nicotine, the steady-state current—voltage relationship for the nicotine-induced inward current was measured and compared with the curve for CCh, which showed no appreciable channel blocking activity. The current—voltage curves for 12.5 μM CCh and 2.5 μM nicotine were virtually superimposable, with no sign of the relative reduction in steady-state current that would be expected if nicotine had channel-blocking activity. Small, but significant differences in the relative amplitudes of the two kinetic components were observed in the spectra of noise evoked by different agonists. No measurable dependence of the relative sizes of the two components on agonist concentration or membrane potential was observed. The evidence supports the hypothesis that the two kinetic components are the product of two independent channel populations for which the agonists tested show some slight differences in selectivity. (Auth. Abs. Mod.)

84–1662. Grygiel, J. J.; Miners, J. O.; Drew, R.; Birkett, D. J. **Differential Effects of Cimetidine on Theophylline Metabolic Pathways.** *European Journal of Clinical Pharmacology* 26(3): 335-340, April 1984.

The effects of cimetidine (1 g/day) on theophylline disposition and metabolism were examined in four smokers (≥ 20 cigarettes/day) and four nonsmokers for single dose intravenous and chronic oral administration of theophylline. In the intravenous study the effect of cimetidine on plasma theophylline clearance was more marked in smokers (22.7 percent reduction) than in nonsmokers (12.2 percent reduction). In the multiple dose study the effect of cimetidine on theophylline clearance was also greater in smokers (28.3 percent decrease) than in nonsmokers (11.3 percent decrease). Reduction in clearance was largely due to a reduction in metabolic clearances by 3-demethylation (Cl_{3DM}) and 1-demethylation (Cl_{1DM}) with no significant effect on clearance by 8-oxidation. There was a strong correlation between Cl_{3DM} and Cl_{1DM} ($r=0.98$, $p<0.001$) in both control and cimetidine study phases, whereas other correlations between partial clearances were less marked and were not apparent during the cimetidine phase. Results are consistent with the view that 1- and 3-demethylation of theophylline are carried out by a common form of cytochrome P-450 that is selectively induced by cigarette smoking and preferentially inhibited by cimetidine. (Auth. Abs.)

84–1663. Hall, S. M.; Herning, R. I.; Jones, R. T.; Benowitz, N. L.; Jacob, P., III. **Blood Cotinine Levels as Indicators of Smoking Treatment Outcome.** *Clinical Pharmacology and Therapeutics* 35(6): 810-814, June 1984.

Pretreatment blood cotinine levels, expired carbon monoxide levels, and self-reported cigarette consumption were evaluated as predictors of smoking treatment outcome in 114 patients. Blood cotinine level was the most useful predictor of nicotine intake. High blood cotinine level was the best predictor of dropping out before completion of treatment and of smoking at the end of treatment and at followups. While cotinine level apparently has good predictive power with respect to variables of smoking cessation outcome, it appears to explain at most 12 percent of the variance observed in abstinence status. Other variables selected from both psychologic and physical domains are also important and not yet well defined. In many settings, cotinine levels are both expensive and difficult to determine; subjective self-report measures are inexpensive and easy to obtain. Whether the increment in explained variance is worth the additional cost will depend in part on the nature of the research or treatment question. (Auth. Abs. Mod.)

84–1664. Hayashi, E.; Isogai, M.; Kagawa, Y.; Takayanagi, N.; Yamada, S. **Neosurugatoxin, a Specific Antagonist of Nicotinic Acetylcholine Receptors.** *Journal of Neurochemistry* 42(5): 1491-1494, May 1984.

Neosurugatoxin (NSTX) (3 to 30 nmol), recently isolated from the Japanese ivory mollusk (*Babylonia japonica*), exerted potent antinicotinic action in isolated guinea pig ileum. Dose—response curves for nicotine-induced contraction of ileum were shifted to the right and depressed gradually as the concentration of NSTX was increased. Specific (^{3}H)nicotine binding to rat forebrain membranes was saturable, reversible, and of high affinity. Nicotinic cholinergic agonists exhibited a markedly greater affinity for (^{3}H)nicotine binding sites than a muscarinic agonist, oxotremorine. Although α-bungarotoxin had no effect on (^{3}H)nicotine binding, low concentrations (1 nmol to 1 μmol) of NSTX inhibited (^{3}H)nicotine binding in the forebrain membranes; IC_{50} value, 69 ± 6 nmol. However, NSTX did not affect muscarinic receptor binding in the brain. Data indicate that NSTX may be of interest as a neurotoxin with a selective affinity for ganglionic nicotinic receptors. (Auth. Abs. Mod.)

84–1665. Heath, C. W., Jr.; Nadel, M. R.; Zack, M. M., Jr.; Chen, A.T.L.; Bender, M. A.; Preston, R. J. **Cytogenetic Findings in Persons Living Near the Love Canal.** *Journal of the American Medical Association* 251(11): 1437-1440, March 16, 1984.

Analysis of peripheral blood from 46 residents of Love Canal, a former dump site for chemical wastes in Niagara Falls, showed that cigarette smoking was associated with an increase in sister chromatid exchange frequency. Participants included 17 persons in whom cytogenetic analyses had been performed in 1980 and 29 persons who, in 1978, had been living in seven homes that directly adjoined the canal and in which environmental tests showed elevated levels of chemicals spreading from the canal. The frequency of chromosomal aberrations did not differ significantly from controls, although males had more symmetrical translocations ($p=0.02$) and a higher proportion of rings and dicentrics with associated fragments ($p=0.05$) than females, while females had more supernumerary, apparent acentrics than males ($p=0.01$). Smokers showed an increase in sister chromatid exchange frequency and a lower proportion of rings and dicentrics with associated fragments than nonsmokers ($p=0.01$). No karyotype abnormalities were found. (Auth. Abs. Mod.)

84–1666. Hoover, K. L. (Editor). **Use of Small Fish Species in Carcinogenicity Testing.** U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, 419 pp.

Benzo(*a*)pyrene, 7,12-dibenz(*a*)anthracene, and diethylnitrosamine are among carcinogens discussed in the proceedings of an international symposium on carcinogenicity testing using small fish in a multitiered testing scheme and as monitors of environmental water pollution. Topics include (1) proposed species for in vivo testing, (2) alternative testing systems using fish, (3) maintenance of fish in the laboratory, (4) special problems and topics related to use of fish in cancer research, and (5) metabolic handling of xenobiotics in fish. See also 84-1646, 84-1671, 84-1675, 84-1687, 84-1688, 84-1705.

84–1667. Hukkelhoven, M.W.A.C.; Dijkstra, A. C.; Vermorken, A.J.M. **Human Hair Follicles and Cultured Hair Follicle Keratinocytes as Indicators for Individual Differences in Carcinogen Metabolism.** *Archives of Toxicology* 53(4): 265-274, August 1983.

Benzo(*a*)pyrene (BaP) metabolism in freshly isolated human hair follicles, cultured hair follicle keratinocytes, and cells cultured from human bronchial epithelium was analyzed by high performance liquid chromatography. All tissue types contained comparable quantities of the most important organic solvent-soluble metabolites: 9,10-dihydrodiol-BaP, 7,8-dihydrodiol-BaP, quinones, and phenols. Two early eluting compounds were also detected. Water-soluble metabolites were quantitatively unimportant in both types of cultured cells and appeared to be primarily glucuronide and sulfate conjugates with the monohydroxides and the 7,8-dihydrodiol of BaP. This metabolic pattern is compared with that of monocytes and lymphocytes that have been used frequently in population studies and with data from other types of human epithelial cells. It is concluded that human hair follicles and cultured keratinocytes from these organs can be useful for detection of individual differences in carcinogen metabolism. (Auth. Abs.)

84–1668. Jones, C. A.; Santella, R. M.; Huberman, E.; Selkirk, J. K.; Grunberger, D. **Cell Specific Activation of Benzo(*a*)pyrene by Fibroblasts and Hepatocytes.** *Carcinogenesis* 4(11): 1351-1357, 1983.

The cell-specific activation of benzo(*a*)pyrene (BaP) by embryonic fibroblasts and by mature hepatocytes to intermediates that can interact with DNA or cause mutations in Chinese hamster V79 cells was investigated. At concentrations of up to 15 μM, BaP was activated to mutagenic intermediates for the V79 cells by embryonic fibroblasts but not by hepatocytes. However, hepatocytes from rats that had been pretreated with an inducer of the mixed function oxidases, 3-methylcholanthrene, did metabolize higher doses of BaP (>15 μM) to mutagenic intermediates. BaP was extensively metabolized by both cell types, but the hepatocytes and fibroblasts showed differences in both the profiles of BaP metabolites and the nature of the BaP-DNA adducts formed. Results suggest that the differences in the response of embryonic fibroblasts and mature hepatocytes in the activation of BaP to a mutagen for mammalian cells is determined at least in part by the overall balance of oxidation and detoxification processes in the cells and, hence, by the levels of critical oxidative intermediates that interact with DNA. (Auth. Abs. Mod.)

84–1669. Kazantzis, G.; Armstrong, B. G. **Renal Function in Relation to Low Levels of Cadmium Exposure in a Group of Smelter Workers.** *Environmental Health Perspectives* 54: 193-199, March 1984.

Smokers had significantly higher mean urine cadmium (Cd-U) levels than nonsmokers, but no differences were noted between ex-smokers and never smokers. Blood and urine samples were obtained from 274 smelter workers (50 percent smokers) and urine samples from 48 controls (35 percent smokers). Cadmium β_2-microglobulin and creatinine were estimated in blood and urine, and total protein in urine. Mean concentrations of Cd-U were as follows: nonsmoking controls 0.53±14 percent, smoking controls 0.91±11 percent, nonsmoking smelter workers 1.46±11 percent, and smoking smelter workers 2.46±6 percent nmole/mmole creatinine. It is noted that smoking-related differences were greater in exposed workers than in controls, suggesting an interaction between industrial exposure and cigarette use. Blood Cd levels showed a similar pattern. Mean β_2-microglobulin in urine was not significantly different in smelter workers and controls. Mean total protein in urine was 20 percent higher in smelter workers (p approximately 0.01). Small but significant positive correlation coefficients were observed between cadmium in urine and β_2-microglobulin in urine ($r=0.13$), total protein in urine ($r=0.23$), and β_2-microglobulin clearance ($r=0.15$), although these may be artifactual. (Auth. Abs. Mod.)

84–1670. Xim, H. R.; Kim, C. K.; Kim, D. S.; Hahn, S. **Heupyon & Nicotini Paekhyolku Yomseke Michinun Yonghyange Kwar Han Yonku. [Studies on the Effect of Nicotine and Cigarette Smoke in Human Leukocyte Chromosomes.]** *Yonsei Journal of Medical Science* 14(2): 93-106, 1981, Korean.

Results showed that frequencies of sister chromatid exchanges (SCE) were higher in 13 smokers than in 10 healthy nonsmokers, and increased according to number of cigarettes smoked/day. Age, sex, and duration of smoking did not affect SCE rates. Blood specimens were obtained and examined for chromosome aberrations and SCE. In a related study to determine whether exposure of human leukocytes to cigarette smoke condensate (CSC) and nicotine would lead to inhibition of mitosis, increase in SCE, and chromosome aberrations, leukocyte cultures from one smoker and one nonsmoker were used. Nicotine concentrations of 10, 20, 30, 40, and 50 μg/ml and CSC of 0.25, 0.5, 1.0, 1.5, and 2.0 mg/ml were prepared. In both nicotine- and CSC-treated leukocytes, there were decreased mitotic indices when concentrations were increased. Increased chromosome aberrations and SCE were found in CSC-treated leukocytes, but not in leukocytes treated with nicotine.

84–1671. Klaunig, J. E. **Establishment of Fish Hepatocyte Cultures for Use in In Vitro Carcinogenicity Studies.** In: Hoover, K. L. (Editor). *Use of Small Fish Species in Carcinogenicity Testing.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, pp. 163-173.

Unscheduled DNA synthesis (UDS) was induced in catfish and trout hepatocytes after exposure to dimethylnitrosamine, aflatoxin B_1, benzo(*a*)pyrene, and N-methyl-N'-nitro-N-nitrosoguanidine (MNNG). Trout hepatocytes displayed a decrease in UDS induction with aflatoxin B_1 with increased age of the cultures. However, UDS induced by MNNG remained constant throughout the culture period. Methods were developed for the isolation and primary culture of rainbow trout (*Salmo gairdneri*) and channel catfish (*Ictalurus punctatus*) liver cells. Using a two-step perfusion technique, an average of 2.75 and 2.87 liver cells/g body weight was isolated from the trout and catfish, respectively. Hepatocytes represented 91.4 percent (trout) and 90.1 percent (catfish) of the isolated cells. Both catfish and trout hepatocytes displayed a linear decrease in survival with increased duration of culture. DNA synthesis in the hepatocytes showed a similar decrease. Approximately 2.8 percent (trout) and 3.5 percent (catfish) of the hepatocytes exhibited nuclear labeling with (^{3}H)dThd immediately after isolation. The labeling index decreased to zero by the 10th day of culture. Cytochrome P-450 and B-5 activity initially declined rapidly for both trout and catfish hepatocytes after placement in culture; however, these activities leveled off at low but measurable values for the first 8 days of culture. (Auth. Abs. Mod.)

84–1672. Koley, J.; Saha, J. K.; Koley, B. N. **Positive Inotropic Effect of Nicotine on Electrically Evoked Contraction of Isolated Toad Ventricle.** *Archives Internationales de Pharmacodynamie et de Therapie* 267(2): 269-278, February 1984.

Nicotine produced a concentration-dependent positive inotropic effect on electrically evoked contractions of isolated spiral-cut toad ventricle. Prior administration of propranolol, hexamethonium, hemicholinium-3, or guanethidine failed to antagonize the nicotine effect, thus ruling out mediation through activation of beta-adrenoceptors, ganglionic activation, presynaptic liberation of acetylcholine, and liberation of norepinephrine from the sympathetic nerve endings, respectively. The positive inotropic effect is probably mediated through mobilization of calcium, since prior incubation with verapamil, EDTA (which chelates the extracellular calcium), and calcium-free Ringer's solution prevented the effect. Results suggest that the positive inotropic effect of nicotine may be due to the facilitating effects of nicotine on calcium ion exchange or mobilization of membrane-bound calcium ions. (Auth. Abs. Mod.)

84–1673. Kulkarni, M. S.; Anderson, M. W. **Persistence of Benzo(*a*)pyrene Metabolite:DNA Adducts in Lung and Liver of Mice.** *Cancer Research* 44(1): 97-101, January 1984.

Following p.o. administration of (^{3}H)-benzo(*a*)pyrene ((^{3}H)-BaP, 6 mg/mouse) in A/HeJ and C57BL/6J mice, animals were killed at times ranging from 10 hours to 28 days and BaP metabolite:DNA adducts were analyzed by high-pressure liquid chromatography. The major adduct identified was the (+)-7β-8α-dihydroxy-9α,10α-epoxy-7,8,9,10-tetrahydro-BaP:deoxyguanosine adduct. A 7β,8α-dihydroxy-9β,10β, epoxy7,8,9,10-tetrahydro-BaP:deoxyguanosine adduct and an unidentified adduct were also observed. The disappearance of (+)-7β,8α-dihydroxy-9α, 10α-epoxy-7,8,9,10-tetrahydro-BaP adduct in A/HeJ mice followed first-order kinetics over the time period examined, with a half-life of 18 and 9 days in lung and liver, respectively. Decay in C57BL/6J mice was biphasic in both tissues. Data on cell turnover suggest that there is active removal of adducts in liver, but that normal DNA turnover can account for the partial or possibly total observed disappearance of adducts in lung. Results suggest that tissue specificity for BaP-induced neoplasia in A/HeJ mice may be related to the relative persistence of adducts and high cell turnover rates in lung. In contrast, results on formation and persistence of adducts and cell turnover do not provide an explanation for the strain difference in susceptibility to BaP-induced pulmonary adenomas. Rates of removal of adducts in A/HeJ mice were not significantly different at a 500-fold lower BaP dose. (Auth. Abs. Mod.)

84–1674. Lederer, J. **Cigarettes, Contraceptifs et Lipidogenese Chez la Femme. [Cigarette Smoking, Oral Contraceptives and Lipidogenesis in Women.]** *Semaine des Hopitaux* 59(49): 3413-3416, December 29, 1983, French.

In 104 women (18 to 45 years of age) studied, 27 used oral contraceptives, 22 smoked, and 26 did both; 29 who used neither served as controls. Triglycerides were significantly higher and high density lipoprotein (HDL) cholesterol was significantly lower in women who used oral contraceptives or who smoked than in controls. A cumulative effect was found in women who smoked and used oral contraceptives. Triglyceride levels in oral contraceptive users were 82.7±7.8; in smokers, 93.4±8.8; and in those who used both, 118.4±10.2. HDL cholesterol levels were 51.3±3.9, 44.2±3.8, and 42.4±3.8, respectively.

84–1675. Melius, P. **Comparative Benzo(*a*)pyrene Metabolite Patterns in Fish and Rodents.** In: Hoover, K. L. (Editor). *Use of Small Fish Species in Carcinogenicity Testing.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, pp. 387-390.

Benzo(*a*)pyrene (BaP) is converted to 3- hydroxybenzo(*a*)pyrene, 9-hydroxybenzo(*a*)pyrene, BaP-4,5- dihydrodiol, BaP-7,8-dihydrodiol, BaP-9,10-dihydrodiol, and BaP quinones by postmitochondrial supernatant or microsomes in such fish as rainbow trout, flounder, salmon, mullet, little skate, *Fundulus grandis*, and sea catfish. It is also now well established that many fish convert BaP to potent mutagenic metabolites, as demonstrated with the Ames test, especially when the fish are induced with Aroclor 1254 or 3-methylcholanthrene. Metabolite patterns obtained at different substrate concentration levels indicate that metabolism is more complex at low concentrations when metabolites are recycled in the in vitro system. (Auth. Abs.)

84–1676. Mikhail, M. M.; El-Dakhakhny, M.; Ghoneim, M. Th. **Effect of Experimental Chronic Exposure to Tobacco Smoke on the Plasma Level of Some Suprarenal Cortical Hormones, Angiotensin II, Serum Sodium and Potassium.** *Bulletin: Alexandria Faculty of Medicine* 17(1): 149-154, March 1981.

Fifteen rabbits (1 to 1.5 kg) were exposed to cigarette smoke in a special apparatus, twice daily (each session about 10 min) for 3 months. Control animals were sham-exposed in the same manner. After 3 months, smoke-exposed rabbits showed a 12.3 percent increase in corticosterone ($p > 0.05$), a 202.8 percent increase in aldosterone ($p < 0.001$), and a 95.3 percent increase in angiotensin II ($p < 0.001$) relative to control animals. Conversely, serum sodium and potassium levels were reduced 3.53 and 6.42 percent ($p > 0.05$), respectively, in treated rabbits compared with controls. The possible mechanisms involved in the increased formation of angiotensin II are described, and a suggested associated role with nicotine and other factors in the development of hypertension is discussed.

84–1677. Miners, J. O.; Attwood, J.; Birkett, D. J. **Determinants of Acetaminophen Metabolism: Effect of Inducers and Inhibitors of Drug Metabolism on Acetaminophen's Metabolic Pathways.** *Clinical Pharmacology and Therapeutics* 35(4): 480-486, April 1984.

Neither cigarette smoking nor cimetidine pretreatment had a significant effect on any of the metabolic pathways of acetaminophen, studied after a single 1 g oral dose in 12 healthy men (6 cigarette smokers) and in 6 men receiving anticonvulsant drugs for epilepsy. Healthy subjects were studied before and after 1 week of pretreatment with cimetidine (1 g/day) or sulfinpyrazone (800 mg/day). There was no significant difference in acetaminophen clearance (Cl_{AP}) between nonsmokers and smokers; cimetidine pretreatment had no effect on Cl_{AP}. In contrast, sulfinpyrazone pretreatment increased Cl_{AP} by 23 percent, while Cl_{AP} was 46 percent greater in epileptic subjects who received anticonvulsant drugs than in controls. In both cases the increase in Cl_{AP} was a result of induction of acetaminophen glucuronidation and oxidation; clearance of glucuronic acid conjugate was 26 percent and 59 percent greater and clearance of glutathione-derived conjugates (reflecting the activity of the oxidative pathway) was 43 and 60 percent greater in groups given sulfinpyrazone and anticonvulsants, respectively. Also, clearance of glutathione conjugates was 41 percent higher in smokers than in nonsmokers, but the increase was not significant. However, in three subjects who consumed 30 to 40 cigarettes/day, clearance was 65 to 85 percent higher, while in those who consumed 15 to 20/day, clearance was similar to that of nonsmokers. (Auth. Abs. Mod.)

84–1678. Nikolin, B.; Lekic, M.; Rashid Mahmud, M. A.; Visnjevac, O. **Investigation of the Content and Ratio of Cotinine and Nicotine-1′-N-Oxide in the Urine of Smokers.** *Arhiv za Higijenu Rada i Toksikologiju* 34(3): 201-207, 1983.

The cotinine/nicotine-1′-N-oxide ratio in the urine of lung and liver cancer patients who smoked was significantly higher than in normal smokers. Nicotine metabolites (cotinine, nicotine-1′-N-oxide, nicotine 1, 1′-N-dioxide, and others) are formed by metabolic degradation of nicotine in smokers. Urine samples were collected from 17 cancer patients (aged 35 to 75, 4 females) and 18 healthy subjects (aged 21 to 50, 3 females), all smokers of 20 to 60 cigarettes/day. Urine content of nicotine, cotinine, and nicotine-1′-N-oxide was analyzed and ratios determined. The urinary ratio of cotinine/nicotine-1′-N-oxide was 7 to 10 times higher in cancer patients. It is suggested that an increased ratio may be an early indicator of neoplastic disease, although whether the change in ratio is a causative mechanism or the result of disease-induced changes in nicotine metabolism is unknown.

84–1679. Nilsen, A. M.; Madslien, O.; Mylius, E. A.; Gullvag. B. M. **Alveolar Macrophages From Expectorate Samples: A Stress Signal From Occupational Pollution.** *Environmental Contamination and Toxicology* 32(5): 517-524, May 1984.

Although workers in some high exposure categories had more alveolar macrophages (AM) in sputum samples than workers with less exposure, smokers had more AM than nonsmokers in all job categories. Sputum samples for workers in a coke plant and an iron works were analyzed and compared with samples from a control group. At the coke plant, a synergistic relationship between cigarette smoke and components in workroom air was noted; components present in larger amounts or only at the top of the oven caused a stronger reaction with smoking than chemicals also present in other working areas. Similar, but not statistically significant, differences were found at the iron works. It is suggested that the number of AM in sputum from workers be used to measure the strain on the lungs related to occupational exposures.

84–1680. Norman, T. R.; Burrows, G. D. **Plasma Concentrations of Benzodiazepines—A Review of Clinical Findings and Implications.** *Progress in Neuro-Psychopharmacology and Biological Psychiatry* 8(1): 115-126, 1984.

A review of the treatment efficacy and pharmacokinetics of benzodiazepines notes that smoking has a significant effect on benzodiazepine pharmacology. Cigarette smoke components are inducers of hepatic drug metabolizing enzymes. Oral clearance is higher and elimination half-life is shorter in smokers. Also, a lower incidence of side effects has been observed among elderly smokers, suggesting a smoking-related lower plasma concentration of the drugs and their metabolites. Other factors influencing benzodiazepine pharmacokinetics include age, sex, disease state, coadministration of other drugs, and alcohol consumption. Also discussed are methods of measuring plasma concentrations, metabolism, and pharmacokinetics of benzodiazepines; new benzodiazepines; anxiolytic responses; and overdosage.

84–1681. Nowicki, P.; Sexton, M.; Hebel, J. R. **Salivary Thiocyanate in Pregnant Smokers: A Comparison of Two Collection Methods.** *Addictive Behaviors* 9(1): 33-39, 1984.

Results showed that thiocyanate measured from saliva samples collected from pregnant women by either the dental roll or the direct method discriminated smokers from nonsmokers. Subjects were 59 pregnant women (34 smokers, 25 nonsmokers) ranging in age from 17 to 35. Smokers were defined as those who smoked at the beginning of pregnancy and were still smoking at the time of the study. The mean number of reported cigarettes smoked/day decreased during pregnancy from 15.6 to 11.7, but smokers were still separated into two groups: light (1-9 cigarettes/day), and heavy (≥ 10 cigarettes/day). Mean thiocyanate for light smokers was 2310.0 µmol/l for the dental roll method and 2437.1 µmol/l for the direct method. For heavy smokers, levels were 3389.0 and 3122.0, respectively. Thus, spitting directly into the collection container resulted in as good a discrimination of smokers from nonsmokers as the more complicated method of having subjects chew a dental roll. Advantages of using the direct method in large research projects are discussed. The salivary data are consistent with earlier observations of higher serum thiocyanate levels during pregnancy, suggesting that thiocyanate may be metabolized differently during this time. (Auth. Abs. Mod.)

84–1682. Pallotti, G.; Consolino, A.; Bencivenga, B.; Iacoponi, V.; Morisi, G.; Taggi, F. **Lead Levels in Whole Blood of an Adult Population Group From Rome.** *Science of the Total Environment* 31(1): 81-87, October 1983.

Smoking habits were positively associated with blood lead (Pb) levels in 801 nonoccupationally exposed adults in Rome. Also, a correlation between percentage of subjects with blood Pb levels higher than 350 µg/l and daily cigarette consumption was verified. Blood Pb levels were also correlated with age and sex, but no associations were found for alcohol consumption, residence, or other variables. Total mean Pb blood level was 173 µg/l (198 µg/l for males and 150 µg/l for females).

84–1683. Rose, J. E. **Discriminability of Nicotine in Tobacco Smoke: Implications for Titration.** *Addictive Behaviors* 9(2): 189-193, 1984.

To determine whether smokers' puff by puff ratings of strength and desirability of smoke would correlate with nicotine delivery and to assess the role of olfaction in nicotine discriminability, 15 male and female smokers (mean age 38.8 years, mean consumption 39.6 cigarettes/day) were presented with puffs from cigarettes with either high (2.5 mg), medium (1.5 mg), or low (0.5 mg) nicotine content. Tar content was controlled by using research cigarettes and a smoke-mixing device that varied only nicotine. Puffs were in random order during two conditions: the first allowed olfactory stimuli to be used in discrimination; the second blocked olfaction by occluding subjects' nostrils. In both conditions, subjects discriminated between the nicotine content of different puffs, with higher nicotine puffs rated as significantly stronger (by roughly 50 percent). Subjective desirability ratings did not vary with nicotine delivery. The implications of the magnitude of change in subjects' ratings for theories of nicotine titration are discussed. (Auth. Abs. Mod.)

84–1684. Rose, J. E.; Zinser, M. C.; Tashkin, D. P.; Newcomb, R.; Ertle, A. **Subjective Response to Cigarette Smoking Following Airway Anesthetization.** *Addictive Behaviors* 9(2): 211-215, 1984.

Contrary to the predictions of a strict pharmacologic hypothesis, results of an experiment to determine whether the blockade of taste and related stimuli by a short-acting topical anesthetic would affect cigarette craving and smoking satisfaction showed that partial blockade of the sensory input from tobacco smoke had a dramatic effect on self-reported cigarette craving. Participants were 7 females and 11 males, average age 31 years, who smoked an average of 24 cigarettes/day. Subjects were given one cigarette to smoke after (1) mouth anesthesia, obtained by rinsing the mouth with 2 percent lidocaine; (2) mouth and pharyngeal anesthesia, in which subjects rinsed and gargled with 2 percent lidocaine; (3) upper and lower airway anesthesia, in which subjects rinsed and gargled with 2 percent lidocaine and inhaled a mist containing 4 percent lidocaine (60 breaths); and (4) saline control (all solutions saline). A significant linear decline in cigarette craving occurred with increasing anesthesia, and desirability ratings over the first several puffs were also reduced by anesthesia. Results may have important clinical implications. Although the central effects of nicotine were not blocked and only a portion of the sensory effects of the smoke was eliminated, blunting of sensory feedback was sufficient to reduce craving, raising new possibilities for smoking cessation strategies and demonstrating that local anesthetics can be useful tools for evaluating the relative importance of sensory vs. pharmacologic factors in smoking motivation. (Auth. Abs. Mod.)

84–1685. Saiani, L.; Kageyama, H.; Conti-Tronconi, B. M.; Guidotti, A. **Purification and Characterization of a Bungarotoxin Polypeptide Which Blocks Nicotinic Receptor Function in Primary Culture of Adrenal Chromaffin Cells.** *Molecular Pharmacology* 25(2): 327-334, March 1984.

The nicotine- and acetylcholine (ACh)-induced release of endogenous catecholamines in cultured bovine chromaffin cells was inhibited by commercial preparations of α-bungarotoxin. However, the potency of different lots of α-bungarotoxin was not related to the α-bungarotoxin peptide content, but to that of another peptide (termed P-4 bungarotoxin) present as an impurity in the α-bungarotoxin preparations. P-4 Bungarotoxin was isolated and purified to homogeneity by high pressure liquid chromatography (HPLC). The purified P-4 bungarotoxin contained approximately 121 amino acid residues, and differed in amino composition, molecular weight, and amino acid sequence from α-bungarotoxin and β-bungarotoxin. P-4 Bungarotoxin blocked ACh-induced release of endogenous catecholamines, but failed to block potassium chloride-induced catecholamine release. Although P-4 bungarotoxin showed phospholipase A_2 activity, its effect on ACh-evoked catecholamine release persisted when the phospholipase activity was blocked (99.9 percent) by treatment of the toxin with *p*-bromophenacyl bromide. P-4 Bungarotoxin may represent a useful tool with which to study nicotinic receptor function in sympathetic and central nervous system neurons. (Auth. Abs. Mod.)

84–1686. Sangster, B.; De Groot, G.; Loeber, J. G.; Derks, H.J.G.M.; Krajnc, E. I.; Savelkoul, T.J.F. **Urinary Excretion of Cadmium, Protein, Beta-2-Microglobulin and Glucose in Individuals Living in a Cadmium-Polluted Area.** *Human Toxicology* 3(1): 7-21, 1984.

Male smokers living in a cadmium (Cd)-polluted area of Stadskanaal had significantly higher urine levels of protein ($p<0.01$) and glucose ($0.01<p<0.05$) compared with nonsmokers. Urinary excretion of Cd, protein, β-2-microglobulin, and glucose was measured in 289 residents (exposed group) and compared with levels in 293 controls. After stratification by smoking status, age, and sex, analyses revealed (1) in both exposed and control groups, Cd excretion was age dependent and increased more rapidly in females than in males; (2) among male controls, Cd excretion was significantly higher ($p<0.001$) in smokers compared with nonsmokers; (3) in the exposed group, male nonsmokers excreted more Cd ($p=0.05$), protein ($p<0.01$), and glucose ($p<0.01$) than controls. Female nonsmokers in the exposed group excreted more glucose ($0.01<p<0.05$) than controls. Although Cd pollution was associated with increased Cd excretion and a slight difference in renal function in some categories (possibly related to differing Cd body burden), it is concluded that the observed difference does not demonstrate a potential health hazard. (Auth. Abs. Mod.)

84–1687. Schoor, W. P. **Benzo(*a*)pyrene Metabolism in Marine Fish and Some Analytical Aspects of Its Metabolites.** In: Hoover, K. L. (Editor). *Use of Small Fish Species in Carcinogenicity Testing.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, pp. 391-396.

Fish metabolize benzo(*a*)pyrene (BaP) in much the same way as mammals: induction of mixed function oxidase takes place, and metabolites are conjugated for excretion. However, dosages and response times vary between species. To determine species-related differences in BaP metabolism, individual metabolite patterns and conjugate formation must be established under various induction schemes. Recommendations are given for standardizing the analytical biochemistry of induction, incubation, microsomal fractions, conjugation reactions, and BaP metabolite analysis. Technical problems in the analysis of metabolites are discussed. Using BaP as a substrate, data from phenobarbital- and 3-methylcholanthrene-induced mullet and killifish are presented, showing the activities of the mixed function oxidase and transferase systems.

84–1688. Stegeman, J. J.; Woodin, B. R.; Binder, R. L. **Patterns of Benzo(*a*)pyrene Metabolism by Varied Species, Organs, and Developmental Stages of Fish.** In: Hoover, K. L. (Editor). *Use of Small Fish Species in Carcinogenicity Testing.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, pp. 371-377.

Microsomal preparations of liver from adults of several fish species, including scup, winter flounder, and killifish, metabolized the model polycyclic hydrocarbon carcinogen benzo(*a*)pyrene (BaP) with a marked regiospecificity, forming high percentages of benzo-ring derivatives, but particularly BaP-7,8-dihydrodiol and BaP-9,10-dihydrodiol. Identity was established by mass spectrometry of metabolites formed by scup liver microsomes compared with the spectra obtained for authentic *trans*-dihydrodiols. Whereas the benzo-ring dihydrodiols equaled 40 to 60 percent of the total ethyl acetate-soluble metabolites formed by liver microsomes of the various species, the K-region metabolite BaP-4,5-dihydrodiol was formed in small or undetectable amounts. Microsomal preparations of kidney and gill from scup formed BaP-7,8-dihydrodiol and BaP-9,10-dihydrodiol in proportions similar to those seen in liver. Embryonic tissues of killifish metabolized BaP with a regiospecificity similar to that exhibited by adult liver microsomes. Isomeric forms of BaP-7,8-dihydrodiol are potent proximate carcinogens, and activation of BaP to mutagenic and carcinogenic derivatives is associated with metabolism on the benzo ring of BaP. The widespread appearance of regiospecificity for this portion of the BaP molecule may be reflected in structural requirements for metabolism of other hydrocarbons and indicate that fish are useful in the study of various factors that affect tumorigenesis by polycyclic hydrocarbons. (Auth. Abs.)

84–1689. Stich, H. F.; Rosin, M. P.; Vallejera, M. O. **Reduction With Vitamin A and Beta-Carotene Administration of Proportion of Micronucleated Buccal Mucosal Cells in Asian Betel Nut and Tobacco Chewers.** *Lancet* 1(8388): 1204-1206, June 2, 1984.

Supplemental intake of retinol and beta-carotene by 40 rural Filipino betel chewers was associated with a threefold decrease (4.2 percent to 1.4 percent) in the mean proportion of cells with micronuclei (which can be increased tenfold by carcinogenic stimuli). The frequency of micronuclei in cells scraped from inside the human cheek is a measure of chromosome breakage in earlier cell divisions. The diet of betel chewers was supplemented for 3 months with sealed capsules of retinol (100,000 IU/week) and beta-carotene (300,000 IU/week). The proportion of micronuclei decreased in 37 of the 40 subjects. In 11 unsupplemented chewers, the mean proportion of micronuclei did not change (4.3 percent before and 4.8 percent 3 months later). It is suggested that in this population an increase in dietary intake of retinol and/or carotene might reduce the incidence of oral cancer.

84–1690. Stockley, R. A.; Afford, S. C. **Qualitative Studies of Lung Lavage α_1-Proteinase Inhibitor.** *Hoppe-Seylers Zeitschrift fur Physiologische Chemie* 365(4): 503-510, April 1984.

An immunoelectrophoretic method is described that enables identification of the molecular size of α_1-proteinase inhibitor (α_1-PI) in biological fluids. When applied to bronchoalveolar lavage fluids, this technique clearly demonstrates α_1-PI in three molecular forms: the native molecule, a partially proteolyzed form, and in a form suggestive of a complex with enzyme. Samples showing the presence of native α_1-PI inhibited more porcine pancreatic elastase than samples where no native α_1-PI was seen or where the predominant form was partially proteolyzed α_1-PI ($p<0.01$). Although the predominant band of α_1-PI was more frequently the partially proteolyzed form in current smokers ($p<0.01$), there was no clear difference in the inhibitory function of α_1-PI between current smokers and nonsmokers and those with and those without airflow obstruction. (Auth. Abs.)

84–1691. Sydor, W., Jr.; Lewis, K. F.; Yang, C. S. **Effects of Butylated Hydroxyanisole on the Metabolism of Benzo(*a*)pyrene by Mouse Lung Microsomes.** *Cancer Research* 44(1): 134-138, January 1984.

Butylated hydroxyanisole (BHA) is a commonly used food additive with demonstrated inhibitory action against chemical carcinogenesis in animals. To elucidate the mechanism of anticarcinogenic action, the effects of BHA on benzo(*a*)pyrene (BaP) metabolism were studied with lung microsomes from female mice. BHA treatment (0.5 percent in the diet for 7 days) inhibited BaP metabolism and altered the ratios among different metabolites as analyzed by high performance liquid chromatography. Treatment reduced metabolic formation of 9,10-dihydroxy-9,10-dihydrobenzo(*a*)pyrene, but not production of 3-hydroxybenzo(*a*)pyrene and *trans*-4,5-dihydroxy-4,5-dihydrobenzo(*a*)pyrene. Because the gross microsomal cytochrome P-450 content was not significantly affected, the change of regioselectivity in BaP metabolism was probably due to the alteration of cytochrome P-450 isozyme composition by dietary BHA. General and regioselective inhibition of BaP metabolism were also observed when BHA were added to the lung microsomal incubation mixture. Since the diol-epoxide is a suspected ultimate carcinogen, the observed inhibitions of BaP metabolism in the formation of diol-epoxides may be closely related to the anticarcinogenic action of BHA. (Auth. Abs. Mod.)

84–1692. Wager-Srdar, S. A.; Levine, A. S.; Morley, J. E.; Hoidal, J. R.; Niewoehner, D. E. **Effects of Cigarette Smoke and Nicotine on Feeding and Energy.** *Physiology and Behavior* 32(3): 389-395, March 1984.

Chronic smoke exposure slightly suppressed growth rate and food intake after 14 days in rats and hamsters in a study of the effects of cigarette smoke and nicotine on food intake, weight gain, resting energy output, brown fat mass, and opiate binding (opiates initiate feeding in sated rats). Blood glucose levels and intrascapular brown adipose mass were increased as a result of smoke exposure. Hamsters chronically exposed to cigarette smoke showed decreased body weight; however, food intake was not significantly suppressed. Short-term (5-day) exposure to nicotine (4 and 2 mg/kg/day) suppressed growth rate and food intake. Nicotine (4 and 2 mg/kg) significantly suppressed water ingestion in water-deprived rats and altered the quantities of flavored solutions ingested by rats compared with those ingested by rats receiving no nicotine. Thus cigarette smoke and nicotine exposure affects food intake, energy utilization, and taste perception, all parameters that contribute to overall body mass. However, these parameters vary in a complex manner with only small changes occurring at specific time intervals. (Auth. Abs. Mod.)

84–1693. Wald, N. J.; Idle, M.; Boreham, J.; Bailey, A.; Van Vunakis, H. **Urinary Nicotine Concentrations in Cigarette and Pipe Smokers.** *Thorax* 39(5): 365-368, May 1984.

Urinary concentrations of nicotine were studied in 27 male nonsmokers, 145 men who smoked cigarettes only, and 48 who smoked pipes only. The median urinary nicotine concentrations were <50 ng/ml (the detection limit of the assay for urine tests) in nonsmokers, 1,393 ng/ml in cigarette smokers, and

1,048 ng/ml in pipe smokers. Values were standardized for urinary pH and creatinine concentration, since nicotine excretion is influenced by the acidity of the urine and by urinary flow rate. The high urinary nicotine concentrations in pipe and cigarette smokers indicated that both have relatively high systemic nicotine concentrations. This observation, together with the fact that large prospective studies have shown that pipe smokers have no material excess risk of coronary heart disease whereas cigarette smokers do, provides evidence that nicotine is unlikely to be the major cause of the excess deaths from coronary heart disease in cigarette smokers. This conclusion is consistent with earlier observations based on serum cotinine concentrations in smokers and nonsmokers. (Auth. Abs.)

84–1694. Wood, A. W.; Chang, R. L.; Levin, W.; Yagi, H.; Thakker, D. R.; Van Bladeren, P. J.; Jerina, D. M.; Conney, A. H. **Mutagenicity of the Enantiomers of the Diastereomeric Bay-Region Benz(*a*)anthracene 3,4-Diol-1,2-Epoxides in Bacterial and Mammalian Cells.** *Cancer Research* 43(12, Part 1): 5821-5825, December 1983.

The mutagenicity of four bay-region diol-epoxide isomers of benz(*a*)anthracene was investigated in two histidine-dependent strains of *Salmonella typhimurium* (TA 98 and TA 100) and in Chinese hamster V79 cells. In strain TA 98 the (-)-diol-epoxide 2 (with the hydroxyl group and epoxide oxygen *trans*) was the most active isomer, although there was less than a threefold difference in the mutagenicity of the four diol-epoxides. In strain TA 100, the (+)-diol-epoxide was the most active, and the two isomers with (3S,4R) absolute configuration were three to eight times more active than the two with (3R,4S). The highest degree of sensitivity to absolute configuration was observed in the V79 cells, in which the (+)-diol-epoxide 2 isomer was from 3 to 20 times more mutagenic than the others. The latter isomer has the same absolute configuration as the bay-region diol-epoxide isomers of benzo(*a*)pyrene and chrysene, which are exceptionally mutagenic to mammalian cells and highly tumorigenic in mice. It is concluded that the presence and absolute configuration of the hydroxyl groups and the absolute configuration of the epoxide moiety play important roles in determining mutagenicity, and that stereoselectivity in metabolism and cellular interactions is important for the expression of biological activity of polycyclic aromatic hydrocarbons.

84–1695. Wyss, A.; V[illegible]rca, G. D.; Schnebli, H. P. **Cigarette Smoke Components Are Not Very Effective in Directly Inactivating α_1-Proteinase Inhibitor.** *Hoppe-Seylers Zeitschrift fur Physiologische Chemie* 365(4): 511-516, April 1984.

Whole cigarette smoke was found to only partially inactivate α_1-proteinase inhibitor (α_1-PI) in aqueous solution, although a significant, time-dependent inactivation of α_1-PI by a dimethyl sulfoxide extract of cigarette smoke condensate was observed. Inactivation could be only partially prevented by antioxidants, indicating that it is not exclusively due to oxidation. Thus, inactive α_1-PI found in lung lavage fluids from smokers is probably generated through endogenous mechanisms and not directly through smoke components. (Auth. Abs. Mod.)

See also, **84–1705, 84–1806, 84–1817**

MORTALITY AND MORBIDITY

84–1696. Brandt, E. N., Jr. **Infant Mortality—A Progress Report.** *Public Health Reports* 99(3): 284-288, May-June 1984.

While infant mortality in the United States continues to decline (provisional rate for 1983, 10.9 deaths/1,000 live births, is the lowest in history), the rate of low birth weight infants shows a much slower decline. It is recognized that no single factor (maternal smoking and alcohol use, demographic characteristics, medical and obstetric history, physical stature, nutrition) explains variations in birth weight. Mature, married, college-educated black women who receive prenatal care are still twice as likely as their white counterparts to deliver low birth weight infants. Single-focused approaches will therefore not realize a significant reduction in the black—white gap. Basic principles for further reducing infant mortality and preventing low birth weight include (1) optimal maternal health and control of diseases such as diabetes, hypertension, and anemia; (2) maternal avoidance of behaviors (such as smoking) known to be related to untoward pregnancy outcomes; and (3) optimal maternal age and interval between pregnancies. Federal efforts toward meeting 1990 goals are outlined, noting that many communities and state agencies are also involved in efforts to reduce infant mortality.

84–1697. Marmot, M. G.; Shipley, M. J.; Rose, G. **Inequalities in Death—Specific Explanations of a General Pattern?** *Lancet* 1(8384): 1003-1006, May 5, 1984.

Variation in smoking habits accounted for most, but not all the differences in mortality in the Whitehall study of 17,530 civil servants. All subjects were classified by employment grade and mortality recorded over 10 years. There was a steep inverse relation between grade and mortality. Compared with the highest grade (administrators), men in the lowest grade had three times the mortality rate from coronary heart disease, from a range of other causes, and from all causes combined.

This is larger than differences in mortality nationally between classes I and V. Smoking and other coronary risk factors are more common in the lowest grades, but these differences account for only part of the mortality difference. Also, classifying smokers according to age at onset of cigarette use or tar content of brand consumed did not account for the higher lung cancer mortality in the lower grades. The inverse relation between employment grade and mortality suggests that factors operating from early life may influence adult death rates. (Auth. Abs. Mod.)

See also, **84–1791, 84–1792**

NEOPLASTIC DISEASES

84–1698. Acheson, E. D.; Gardner, M. J.; Winter, P. D.; Bennett, C. **Cancer in a Factory Using Amosite Asbestos.** *International Journal of Epidemiology* 13(1): 3-10, March 1984.

Analysis of mortality data from a cohort of male insulation board workers (n=5,969, >40 percent born after 1940), 4,820 of whom worked with amosite asbestos, showed a greater than additive, but less than multiplicative, interaction between smoking and amosite asbestos exposure. By the end of 1980, 57 lung cancer deaths were observed where only 29 were expected; the excess is present in men entering the factory before and after 1960. Smoking data, available for 2,461, showed 1,883 smokers, 113 ex-smokers, and 465 nonsmokers. There was a slight increase in the proportion of smokers with increasing exposure, and detectable excess risk of lung cancer was limited to current smokers exposed to higher levels of asbestos. Only one lung cancer death occurred among nonsmokers. Apart from five deaths from mesothelioma, no statistically large or significant excesses of mortality from cancers of other sites occurred, but further followup of the cohort is in progress. Nine deaths from asbestosis were recorded. (Auth. Abs. Mod.)

84–1699. Antman, K. H.; Pomfret, E. A.; Aisner, J.; MacIntyre, J.; Osteen, R. T.; Greenberger, J. S. **Peritoneal Mesothelioma: Natural History and Response to Chemotherapy.** *Journal of Clinical Oncology* 1(6): 386-391, June 1983.

Data indicated a trend toward shorter survival among smokers (43 percent of 23 malignant peritoneal mesothelioma patients) and those presenting with ascites in an assessment of the natural history of the disease and efficacy of current treatment. Asbestos exposure was reported by 57 percent of the patients, who were seen at the Dana Farber Cancer Institute and the University of Maryland Cancer Center from 1968 to 1982. Of 18 patients receiving a doxorubicin-containing regimen, 14 had measurable or evaluable disease. One complete response, four partial responses, and one regression (in a patient with evaluable but not measurable disease) were observed, ranging in duration from 6 to 36 months. A single patient remained free of disease for more than 36 months after subsequent radiotherapy. Significant clotting abnormalities (including disseminated intravascular coagulation, massive thrombosis, fatal pulmonary emboli, Coombs-positive hemolytic anemia, and phlebitis) occurred in 22 percent. (Auth. Abs. Mod.)

84–1700. Bewtra, C. **Multiple Primary Bronchogenic Carcinomas, With a Review of the Literature.** *Journal of Surgical Oncology* 25(3): 207-213, March 1984.

Four cases of dual, primary lung cancer are presented, noting that all patients were male, over 60 years of age, and heavy smokers. The average time between the metachronous tumors was 20 months (one case was synchronous). All were different histological types, most commonly a combination of squamous cell carcinoma and adenocarcinomas. Diagnostic criteria, other epidemiological factors, and prognosis are also discussed. Data from the literature review show a striking uniformity in all studies; most, if not all, patients appear to be heavy smokers. One study concluded that when the smoking habits of patients with single primary lung cancer were compared with those with multiple primaries, the only significantly different factor was the number of cigarettes smoked/day.

84–1701. Brinton, L. A.; Blot, W. J.; Becker, J. A.; Winn, D. M.; Browder, J. P.; Farmer, J. C., Jr.; Fraumeni, J. F., Jr. **A Case-Control Study of Cancers of the Nasal Cavity and Paranasal Sinuses.** *American Journal of Epidemiology* 119(6): 896-906, June 1984.

Cigarette smokers (n=93) were found to have a twofold to threefold excess risk of nasal cancer in a case—control study of smoking, occupation, and other risk factors among 160 patients admitted to four hospitals in North Carolina and Virginia between 1970 and 1980, and 290 controls. There was no evidence that alcohol consumption affected risk. Risk in smokers was predominantly for squamous cell tumors. Evidence was found of an elevated risk associated with snuff use, but no increased risks were associated with cigar smoking or chewing tobacco. Employment in the furniture industry was not associated with squamous cell tumors, but did increase the risk of nasal adenocarcinoma fivefold. For employees in industries involving possible exposure to wood dust, there was an approximately threefold excess risk of adenocarcinoma. Elevated risks among males were also associated with occupational exposures to chromates (relative risk (RR) = 5.1) and chemicals (RR = 2.7). Among females, excess risk was associated with employment in the textile industry, particularly for jobs involving dust exposure (RR = 2.3). Elevated risks were also associated with histories of nasal polyps (RR = 8.3), recurrent nosebleeds (RR = 2.0), and sinus problems (RR = 2.7). (Auth. Abs. Mod.)

84–1702. Burch, P.R.J. **Commentary on "40 Years of U.S. Cigarette Smoking and Heart Disease and Cancer Mortality Rates."** *Journal of Chronic Diseases* 37(5): 325-332, 1984.

An analysis of time trends in smoking and mortality from lung cancer and coronary heart disease (CHD), published concurrently in the *Journal of Chronic Diseases*, is criticized because (1) the conclusions are based on mortality data that were age adjusted but not separated by sex; (2) tobacco use was measured as per capita cigarette consumption, ignoring differing smoking rates between men and women; and (3) "risk factors" were equated with "causal factors." Data are presented on a log scale and scatter diagrams plotted separately for CHD and lung cancer mortality. Lung cancer mortality data show changes from one 5-year period to the next that are remarkably synchronous between sexes (although generally larger in men). However, the steep rise in smoking among women lagged 30 years behind that of men, and it is concluded that the large increases in lung cancer mortality are unrelated to cigarette consumption and can be almost entirely accounted for by the near-eradication of tuberculosis. Further, it is noted that the relationship between CHD mortality and cigarette consumption is not linear, nor are the dose—response relationships. Sex- and age-specific trends in cigarette consumption display marked nonuniformity, in contrast with the uniformity in CHD mortality trends. Also, the association between smoking and CHD is dependent on age and sex. It is concluded that the association is largely or wholly noncausal and predominantly constitutional. See also 84-1371.

84–1703. Castonguay, A.; Rivenson, A.; Trushin, N.; Reinhardt, J.; Spathopoulos, S.; Weiss, C. J.; Reiss, B.; Hecht, S. S. **Effects of Chronic Ethanol Consumption on the Metabolism and Carcinogenicity of *N'*-Nitrosonornicotine in F344 Rats.** *Cancer Research* 44(6): 2285-2290, June 1984.

For 4 weeks prior to and during treatment with N'-nitrosonornicotine (NNN), groups of 26 to 30 rats were maintained on either a control liquid diet (groups 1, 3, and 5) or an ethanol-containing liquid diet (groups 2, 4, and 6). One mmol of NNN/rat was injected s.c. (10 mg/kg, groups 3 and 4) three times weekly or added to the liquid diet (17.5 mg/l, groups 5 and 6). The nasal mucosa was the main target tissue of NNN in groups 3 and 4, but both the nasal mucosa and esophagus were major target tissues in groups 5 and 6. In rats treated s.c. with NNN (groups 3 and 4), ethanol consumption had no effect on the distribution and incidence of nasal cavity tumors. In rats treated with NNN added to the control liquid diet or to the ethanol-containing liquid diet, the number of nasal cavity tumors was 18 in group 5 and 26 in group 6 ($p<0.05$). In contrast, the number of rats with esophageal tumors was 25 in group 5 and 20 in group 6 ($p<0.05$). Tissue explants cultured in vitro with NNN showed that the nasal mucosa of ethanol-consuming rats had a 1.5-fold higher ($p<0.05$) α-carbon-hydroxylating activity than control rats. Activating enzymes in the lingual mucosa, esophagus, and liver were not induced by ethanol. Thus the carcinogenic effects of NNN added to an ethanol-containing diet could be due in part to an induction of activating enzymes by ethanol. However, since chronic ethanol consumption had no apparent effect on the incidence of nasal cavity tumors in rats treated by s.c. injection of NNN, factors other than enzyme induction are important in determining the effects of ethanol on NNN carcinogenicity. (Auth. Abs. Mod.)

84–1704. Chan, W. C.; Fung, S. C. **Lung Cancer in Non-Smokers in Hong Kong.** In: Grundmann, E.; Clemmesen, J.; Muir, C. S. (Editors). *Geographical Pathology in Cancer Epidemiology*, Cancer Campaign, Volume 6. New York, Gustav Fischer Verlag, 1982, pp. 199-202.

Among 208 male and 189 female bronchial cancer patients in Hong Kong, 2 males and 84 females were nonsmokers. Interviews with nonsmokers revealed that most were diagnosed after age 70. The most prevalent histological type was adenocarcinoma (44 percent compared with 19 percent squamous and 37 percent others). No association was found between bronchial cancer in nonsmokers and passive smoking or cooking fuels; 40.5 percent of cases, compared with 47.5 percent of matched controls (orthopedic patients), reported passive smoking. It is noted that a high proportion of women were Cantonese (81 percent of cases vs. 70 percent of controls). The potential role of diet is discussed.

84–1705. Consensus Committee. **Summary and Recommendations: A Consensus Report.** In: Hoover, K. L. (Editor). *Use of Small Fish Species in Carcinogenicity Testing.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 65, NIH Publication No. 84-2653, May 1984, pp. 397-404.

An overview of the potential of small fish for use in carcinogenicity testing provides data on various compounds (including benzo(*a*)pyrene (BaP) and nitrosamines) known to induce tumors in fish species. BaP administered in food causes liver neoplasms in rainbow trout, and dimethylnitrosamines in food or water induce tumors in the liver, kidney, swim bladder, and stomach of several species (guppy, medaka, zebra fish, rainbow trout, and others). The advantages to using fish for carcinogenicity assay include their continuous exposure to water-borne chemicals, ease of handling large numbers in the laboratory, low background level of tumor incidence, short latency period, and lower cost. The criteria for species selection involve the ability to thrive on a purified diet, the difficulty of maintaining disease-free states in fish cultures, hardiness in a laboratory environment, accessibility of the species throughout the year, and size. The potential of field studies (applying epidemiological techniques to defined populations in monitored waterways) and requirements for test protocols are also explored. The need for in-depth studies to define the specific physiologic and pathologic effects of carcinogens in fish at the cellular and subcellular level is stressed.

84–1706. Cross, F. T. **Radioactivity in Cigarette Smoke Issue.** *Health Physics* 46(1): 205-208, January 1984.

In an examination of the causal role of radioactivity in tobacco smoke-related lung cancers, it is concluded that the dose of radiation from radon daughters on cigarette smoke particles is insignificant compared with that deposited in the lungs from ambient air, and thus some other factor in smoke must be responsible for its carcinogenicity. Although smoking-related hindrance to mucociliary clearance increases the dose, the change is not dramatic. Further evidence that disintegrating lead and/or polonium are not directly responsible for smoke carcinogenicity includes (1) comparison of lifetime risks of lung cancer between nonsmokers and smokers, (2) comparisons of human and animal studies that did not eliminate radon exposures among controls, (3) incidence of cancer in nonsmoking wives of smokers, (4) data from Newfoundland fluorspar miners, and (5) comparison of ambient ^{210}Po with ^{210}Po produced in the lung by pyrolysis of ^{214}Pb during smoking.

84–1707. Cuzick, J.; Harris, R.; Mortimer, P. S. **Palmar Keratoses and Cancers of the Bladder and Lung.** *Lancet* 1(8376): 530-533, March 10, 1984.

Although there was little useful information about smoking and the prevalence of keratoses in lung cancer patients (all but 1 of the 66 lung cancer patients were smokers), smoking was related to keratoses in 218 hospital controls. Among controls with keratoses, 50 percent were smokers, 38 percent ex-smokers, and 12 percent nonsmokers. Percentages for controls without keratoses were 30, 31, and 39, respectively. Data are from a case—control study of the prevalence of palmar keratoses in 69 bladder cancer patients, the lung cancer patients, and controls. Palmar keratoses were more common in cases, especially among those with cancer of the bladder, than in controls. Among those over 50 years of age, 87 percent of bladder cancer patients and 71 percent of lung cancer patients (but only 36 percent of controls) had one or more keratoses. Prevalence increased with age, and keratoses were more common in males. No features could be found that distinguished the keratoses in cases from those in controls. (Auth. Abs. Mod.)

84–1708. Enstrom, J. E.; Kanim, L. E. **Smoking Cessation Among California Physicians: An Example of Cancer Control.** In: Engstrom, P. S.; Anderson, P. N.; Mortenson, L. E. (Editors). *Advances in Cancer Control: Epidemiology and Research.* Progress in Clinical and Biological Research, Volume 156. New York, Alan R. Liss, Inc., 1984, pp. 255-264.

A 30-year prospective study of 10,130 California physicians, among whom the proportion of smokers declined from 53 percent in 1950 to 10 percent in 1980, is described as an example of cancer control in a defined population. The number of males who smoked in the general population of the United States declined from 53 percent to 38 percent over the same period. The standardized mortality ratio (SMR) for lung cancer among the physicians dropped significantly from 62 in 1950-1959 to 30 in 1970-1979. The SMR for other respiratory, esophageal, and buccal cavity cancers declined from 100 in 1950-1959 to 63 in 1970-1979. Mortality from other smoking-related diseases also declined substantially, but SMRs of other causes of death did not change significantly. It is noted that mortality patterns converged toward those of physicians who were nonsmokers during the entire 30 years. It is estimated that 471 deaths were averted (21 percent of total expected deaths). Of the 446 smoking-related deaths avoided (95 percent of deaths averted), 55 were lung cancer, 13 were other smoking-related cancers, 360 were ischemic heart disease, and 18 were from emphysema, bronchitis, and asthma.

84–1709. Fox, B. H. **Remote Life-Style Causes of Cancer.** *Cancer Detection and Prevention* 7(1): 21-29, 1984.

In a review of cancer risk factors to establish the connection between cancer and intermediate and remote conditions (demographic, economic, political, meteorological, sociocultural), it is noted that tobacco, either alone or with other variables, is a substantial contributor to the total cancer burden. The best estimate of the proportion of all cancer deaths attributable to tobacco is 30 percent. Diet, regarded separately or as part of lifestyle, is another major contributor. In discussing intermediate and remote causes, it is suggested that (1) geography can affect the indigenous food supply and the distribution of various natural protective and harmful substances; (2) poor economic conditions foster undernutrition, while in strong economies, overeating may be a factor; (3) taxation and removing tobacco industry support may be the best ways to reduce consumption; (4) sociocultural conditions such as found in India among betel quid users are related to specific types of cancer and may be difficult to change, although social conditions in the United States since the original Surgeon General's report have had an impact on smoking rates; (5) governmental policies (e.g., raising or lowering the upper permissible carcinogens in products) can have an effect on carcinogenesis; and (6) stress may have differential effects and should be considered a conditional covariate. Classes of preventive action for intermediate and proximal causes of cancer are listed.

84–1710. Fu, H. J.; Gou, J. **Research on Causes of Lung Cancer: Case—Control Study of 523 Cases of Lung Cancer.** *Canadian Journal of Public Health* 75(2): 161-165, March-April 1984.

Data from a case—control mortality study of 523 cases of lung cancer (402 smokers) in three districts of Harbin, China, show that smoking is significantly correlated with lung cancer, chronic bronchitis, and tuberculosis. Information about cases from relatives included general history, occupation, smoking habits, method of house heating, and cooking fuel use. Controls were selected from nonrespiratory deaths matched for age, sex, and district of residence. The risk of lung cancer increases if a person smokes >10 cigarettes/day, started smoking before age 30, and has been smoking >20 years. The risk increases with higher consumption, earlier age of initiation of smoking, and more years as a smoker. Further research is needed to determine if lung cancer risk varies with the consumption of factory-made or handmade cigarettes. Results also showed that the risk of lung cancer increases if a person with chronic bronchitis is a smoker. (Auth. Abs. Mod.)

84–1711. Geddes, D. M.; Spiro, S. G.; Tobias, J. S.; Souhami, R. L.; Harper, P. G. **Lung Cancer: Future Prospects.** In: Duncan, W. (Editor). *Lung Cancer.* Recent Results in Cancer Research, Volume 92. Berlin, Springer-Verlag, 1983, pp. 118-127.

If smoking were to stop today, lung cancer would be a rare disease within 15 years. Incidence is expected to decline in the industrialized countries over the next 20 years. This decline will be accompanied by a progressive shift in age distribution of the disease away from middle life toward the elderly, as the cohort at particularly high risk ages. As a result there may be quite a rapid decline after this cohort has passed through the population, and lung cancer death rates may fall faster than would be predicted from changes in smoking habits. Conversely, in developing countries where smoking is still increasing, a new epidemic of lung cancer can be predicted that will affect predominantly middle-aged men. Screening programs are expensive, unproductive, and unlikely to have widespread application. Advances in the management of lung cancer can be expected with the development of more effective drug therapy and more refined methods of tumor localization, particularly development of monoclonal antibodies, which may result in more precise tumor imaging and improved therapeutic ratios by selectively delivering drugs to the tumor. Nevertheless, the principal challenge remains in prevention rather than treatment. (Auth. Abs. Mod.)

84–1712. Hirsch, J.-M.; Johansson, S. L.; Vahlne, A. **Effect of Snuff and Herpes Simplex Virus-1 on Rat Oral Mucosa: Possible Associations With the Development of Squamous Cell Carcinoma.** *Journal of Oral Pathology* 13(1): 52-62, 1984.

The incidence of malignant tumors was significantly higher in Sprague-Dawley rats exposed to snuff or to snuff and *Herpes simplex* virus type 1 (HSV-1) in combination than in control animals ($p<0.05$). Rats were acutely infected monthly with HSV-1 to simulate recurrence of latent infection. Virus was applied topically to the mucous membrane twice at an interval of 1 month. Snuff was administered between the virus applications and afterward to half the virus-exposed animals. Sham-infected rats were given snuff during the same period (18 months). A fourth group of rats was left untreated. A complete postmortem examination was performed. Two rats exposed to snuff and HSV-1 in combination developed squamous cell carcinoma of the oral cavity. It was also found that rats exposed to snuff alone or in combination with HSV-1 had a higher incidence of tumors or tumor-like conditions than control rats exposed to HSV-1 only. Results indicate that HSV-1 in combination with snuff exposure may be associated with the development of squamous cell carcinomas of the oral cavity. (Auth. Abs. Mod.)

84–1713. Jedrychowski, W. **Interaction of Smoking and Urban Air Pollution in the Etiology of Lung Cancer.** *Neoplasma* 30(5): 603-609, 1983.

Smoking accounted for most excess lung cancer deaths in Cracow, Poland, between 1975 and 1980, although air pollution was a contributing factor. All death certificates (n=33,173) were analyzed and standardized mortality ratios calculated. Lung cancer deaths represented 6.7 percent of all male deaths and 1.6 percent of all female deaths and 32.5 percent of all male cancer deaths and 8.3 percent of all female cancer deaths. Compared with the rest of Poland, lung cancer mortality was 18 percent higher in older males and 42 percent higher in females of all age groups. In Cracow, 66 percent of males and 24 percent of females are smokers, a higher prevalence than in rural Poland. Smokers in Cracow also begin smoking earlier and have a higher daily consumption. Smoking accounted for sex-related differences and most of the urban-rural differences in lung cancer mortality. However, the urban-rural difference was higher in the more heavily polluted areas of Cracow. Analysis of rates by demographic area showed that air pollution contributed significantly to lung cancer mortality in Cracow.

84–1714. Kastenbaum, M. A. **Consistency of Research Data on Passive Smoking and Lung Cancer. (Letter).** *Lancet* 1(8391): 1406, June 23, 1984.

A previous letter (*Lancet* 1(8375): 506, March 3, 1984) is criticized for the suggestion that Garfinkel's reanalysis of the American Cancer Society cohort is consistent with other passive smoking and lung cancer research if adjustment is made for confounding by a proportion of the control group exposed to smoke at work. It is claimed that such a correction would not significantly affect the originally derived risk ratio of 1.2. Further, risk ratios calculated from the suggested corrected rates imply that nonworking wives of smokers are at greater risk than working wives (1.26 vs. 1.13) and working wives of nonsmokers are at greater risk than working wives of smokers (2.05 vs. 1.83), although a daily exposure of 1.82 mg tar at work vs. 0.45 mg tar at home is assumed. Finally, the average crude lung cancer rate for all white females in the United States for the period in question is 8.6, almost the same as the assumed basal rate of 8.7 for lung cancer deaths not associated with passive smoking. See also 84-1078.

84–1715. Koh, H. K.; Sober, A. J.; Day, C. L., Jr.; Lew, R. A.; Fitzpatrick, T. B. **Cigarette Smoking and Malignant Melanoma. Prognostic Implications.** *Cancer* 53(11): 2570-2573, June 1, 1984.

Heavy cigarette smoking (current smoking with a history of >15 pack-years) and 13 other variables were tested for their ability to predict death in 44 heavy smokers and 152 nonsmokers or light smokers, all patients with clinical stage I melanoma. A stepwise proportional hazards general linear model (Cox multivariate analysis) showed that although heavy cigarette smoking as a single variable is an adverse prognostic marker (p=0.0065), it has only suggestive prognostic significance once thickness factors are taken into account (p=0.0747). People who stopped smoking had the same survival as nonsmokers. Clinical stage I patients with melanoma who were heavy smokers presented with thicker lesions than nonsmoking counterparts (p=0.037). Although cigarette smoking may play a role in the biologic behavior of melanoma by mediating an effect on thickness, it need not be considered as an independent stratification criterion when analyzing results of melanoma prognosis or treatment. (Auth. Abs. Mod.)

84–1716. L'Abbe, K. A.; Hoey, J. R. **Cigarette Smoking, Lung Cancer and Canadian Women.** *Canadian Medical Association Journal* 130(12): 1539-1540, June 15, 1984.

Overall female mortality in Canada declined by 16.5 percent between 1970 and 1979, yet lung cancer mortality will double between 1976 and 1987, surpassing breast cancer as the leading cause of cancer-related death in women. Most lung cancers are attributable to smoking. The 1978-1979 Canada Health Survey revealed that 43.8 percent of men and 35.8 percent of women over age 15 are regular smokers, which represents an absolute decrease of 9 percent in men but an absolute increase of 29 percent among women over the past 15 years. The prevalence of smoking in females is approaching that in men and so is the rate of death from lung cancer. Female smokers are increasing in number and are also consuming more cigarettes daily and starting to smoke at an earlier age than older female smokers did. Public policy changes designed to halt the increase, including a ban on all forms of tobacco advertising, are suggested. The ban would be especially effective since more than 8 of 10 women read women's magazines, which accept cigarette ads while publishing very little smoking and health information.

84–1717. Lubin, J. H.; Blot, W. J.; Berrino, F.; Flamant, R.; Gillis, C. R.; Kunze, M.; Schmahl, D.; Visco, G. **Patterns of Lung Cancer Risk According to Type of Cigarette Smoked.** *International Journal of Cancer* 33(5): 569-576, 1984.

Lifelong smokers of nonfiltered cigarettes were at twice the risk of developing lung cancer as lifelong smokers of filtered cigarettes in a case—control study of 7,804 lung cancer patients (6,920 males, 844 females) and 15,2[illegible]7 hospital-based controls. Over 50 hospitals in Austria, France, Germany, Italy, and Scotland participated. After controlling for duration of cigarette use and number smoked/day, the relative risk was 1.7 for males and 2.0 for females. Lung cancer risks for smokers of filtered and nonfiltered cigarettes and for those with a mixed habit (including both filtered and nonfiltered brands) increased in proportion to intensity and duration of smoking and decreased with years since smoking cessation. There was a ninefold excess risk of lung cancer with cigarette use among males, a slightly lower excess for cigarette and pipe smoking (RR=8.1) or cigar (RR=6.9) use, and an even smaller excess risk with exclusive pipe (RR=2.5) or cigar (RR=2.9) use. Among females, almost all smokers used cigarettes exclusively and were at a 3.9-fold excess risk. Findings indicate that prevention activities should continue to emphasize smoking cessation, although switching to low-tar cigarettes may also yield some reduction in lung cancer risk. (Auth. Abs. Mod.)

84–1718. Melamed, M. R.; Flehinger, B. J.; Zaman, M. B.; Heelan, R. T.; Perchick, W. A.; Martini, N. **Screening for Early Lung Cancer. Results of the Memorial Sloan-Kettering Study in New York.** *Chest* 86(1): 44-53, July 1984.

The Memorial Sloan-Kettering lung cancer screening program was begun in 1974 to evaluate sputum cytology as a supplement to the annual chest X-ray examination for early detection and diagnosis. The 10,040 adult male cigarette smokers who enrolled were randomly assigned to annual chest X-ray examination only or a dual screen with annual chest X-ray examination and four monthly sputum cytology evaluations. Over 40 percent of the 288 who developed lung cancer were diagnosed in stage I, and survival was 76 percent at 5 years; overall survival was 35 percent. Nearly one-third of the lung cancers detected on first examination on the dual screen and 14 percent of those on subsequent examinations were found by cytologic examination. The same number of cancers developed in the X-ray screen only group, and were diagnosed at a later date. Despite the delay, survival and mortality were the same, suggesting that the squamous carcinomas detected by cytologic examination alone are very slow growing and tend to remain localized until detectable by X-ray examination. (Auth. Abs.)

84–1719. Mommsen, S.; Aagaard, J.; Sell, A. **An Epidemiological Study of Bladder Cancer in a Predominantly Rural District.** *Acta Chirurgica Scandinavica* 17(3): 307-312, 1983.

Smoking (cigarettes and cigarillos) was a significant risk factor for bladder cancer in a case—control study of 212 patients (165 males, 47 females) and 259 controls matched for age and geographic area. Twenty-five determinants of assumed importance were examined. Bivariate analysis revealed a significantly increased relative risk (RR) of developing bladder cancer associated with cigarette smoking, cigarillo smoking, pipe smoking, tobacco chewing, industrial work, previous venereal disease, work with petroleum or asphalt, consumption of alcohol, and work with oil, gasoline, or chemicals. Cigarette smoking was the strongest independent variable; RR was 2.09

times greater for cigarette smokers than for nonsmokers. RR increased to 4.57 for smokers of both cigarettes and cigarillos. Logistic analysis of the negatively associated determinants revealed nonsmokers and farmers were the weakest independent variables. (Auth. Abs. Mod.)

84–1720. Morrison, A. S.; Buring, J. E.; Verhoek, W. G.; Aoki, K.; Leck, I.; Ohno, Y.; Obata, K. **An International Study of Smoking and Bladder Cancer.** *Journal of Urology* 131(4): 650-654, April 1984.

The effects of cigarette smoking and other forms of tobacco use on the development of bladder cancer were evaluated in 592 cases and 533 controls in Boston, Massachusetts, 553 cases and 731 controls in Manchester, England, and 290 cases and 588 controls in Nagoya, Japan. Cigarette smokers had about twice the incidence of bladder cancer as nonsmokers. In men, the strength of the association between cigarette smoking and bladder cancer was similar in the three areas, but varied somewhat from area to area among women. Bladder cancer risk increased with frequency of cigarette smoking and with deep inhaling. In Boston, men who smoked two or more packs of cigarettes/day and inhaled deeply had nearly seven times the risk of nonsmokers. Ex-smokers had a level of risk between that of current smokers and nonsmokers. However, there was no clear relationship between risk and age at which cigarette smoking began, time since discontinuation among ex-smokers, or use of filters. Overall, there was little difference in bladder cancer risk between men who had and who had not smoked pipes, but pipe smoking did appear to be associated with risk among men who had never smoked cigarettes. Cigar smoking was unrelated to bladder cancer. (Auth. Abs. Mod.)

84–1721. Nielsen, K. O.; Bak-Pedersen, K. **Mucous-Producing Elements in the Laryngeal Mucosa in Smokers With Cancer of the Larynx.** *Cancer* 54(1): 61-64, July 1, 1984.

Morphologic and quantitative studies of goblet cells and mucous glands were done of mucosal biopsies from the subglottis, the entrance to the sinus of Morgagni, and the false vocal cords in 60 smokers with laryngeal cancer, 30 biopsied at diagnosis, the other 30 after radiotherapy. In the nonirradiated group, goblet cell density was increased in the sinus of Morgagni and in the subglottis compared with biopsies from normal nonsmoking controls (n = 20); the increase was significant only in the subglottis. There was a definite tendency to a lower goblet cell density in the irradiated group and an increased number of goblet cell-free areas because of epithelial metaplasia to stratified squamous epithelium, again significant only in the subglottis. In nonirradiated and irradiated smokers, subepithelial mucous glands commonly showed mild to appreciable degenerative changes. This morphologic difference between smokers and nonsmokers may represent the toxic action of tobacco on the laryngeal mucosa and may thus enter into the pathogenesis of laryngeal cancer. (Auth. Abs. Mod.)

84–1722. Nomura, A.M.Y. **Association of Cigarette Smoking and Nitrosamines With Urinary Bladder Cancer: A Review.** In: Henderson, B. E.; Kolonel, L. N.; Thompson, D. J. (Editors). *Third Symposium on Epidemiology and Cancer Registries in the Pacific Basin.* U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Cancer Institute Monograph No. 62, NIH Publication No. 82-2438, January 1983, pp. 185-190.

Several studies have shown a significant positive association between bladder cancer and smoking, with a relative risk of 1.7 to 3.9 in males and 1.3 to 3.8 in females. The relative risk is lower than for lung cancer and smoking, indicating that a population with high smoking prevalence would be expected to have high lung cancer frequency and increased bladder cancer incidence. Among populations with high lung cancer and low bladder incidence are blacks and Pacific Basin Polynesians. To investigate this unexpected finding, metabolic studies are indicated. Several investigators have attempted to measure components of nitrosamines in urine specimens of patients with bladder cancer, cigarette smokers, and controls. Limitations of the studies are discussed, and new studies are proposed if methods of nitrosamine analysis can be improved in the future. (Auth. Abs. Mod.)

84–1723. Nou, E. **The Natural Five-Year Course in Bronchial Carcinoma. Epidemiologic Results.** *Cancer* 53(10): 2211-2216, May 15, 1984.

Of 273 bronchial carcinoma patients (82 percent smokers), 75 percent of cases in smokers were diagnosed by accident compared with 24 percent in nonsmokers. Histologic group distribution also differed: 97 percent of WHO group I (squamous cell), 94 percent of group II (small cell), 57 percent of group III (adenocarcinoma), and 65 percent of group IV (large cell) were found in smokers. Cases, from a 5-year prospective study of the total population of Uppsala County, Sweden, had an observed 5-year survival rate of 7.0 percent. An autopsy rate of 94 percent was achieved, and all diagnoses were histologically or cytologically verified. The 5-year survival rates for squamous cell carcinoma, small cell carcinoma, adenocarcinoma, and large cell carcinoma were 7.5 percent, 0 percent, 11.7 percent, and 3.8 percent, respectively. In 73 percent of all survivors, and in all survivors of adenocarcinoma, the disease was discovered by accident. It is concluded that the intensified search for bronchial carcinoma on chest X-rays taken because of suspicion of other diseases may have contributed to the observed survival gains compared with other studies. The observed "real" 5-year survival rate (including, as survivors, patients dying of other diseases without remaining bronchial carcinoma at autopsy) was 8.8 percent. The corresponding Kaplan-Meier product limit estimated 5-year survival was 7.7 percent. The study is considered to reflect the natural 5-year course of the disease. Results cannot be repeated because of recent advances in therapy. (Auth. Abs. Mod.)

84–1724. Pashcow, M. S.; Mattucci, K. F. **Direct Laryngoscopy: A Retrospective Analysis.** *International Surgery* 68(4): 331-335, October-November-December 1983.

Retrospective analysis of 280 laryngoscopy patients (54 percent males, 46 percent females) showed that tobacco and/or alcohol use was most frequently associated with laryngeal cancer. Data indicated that 62.1 percent of the total group used tobacco, alcohol, or both, but about two-thirds of patients with benign tumors were nonusers. The most common symptom reported was hoarseness (83.6 percent); the most frequent benign and malignant diagnoses were vocal cord polyp and squamous cell carcinoma. Males predominated in all disease entities except vocal cord polyps. Benign disease entities presented most frequently with one or two symptoms, while malignant pathology presented with a varied array and number of symptoms. Most frequently associated with malignancy were "tumor" seen on indirect laryngoscopy, sore throat, dysphagia, otalgia, upper respiratory tract obstruction, hemoptysis, cough, and leukoplakia. Voice abuse occupations were most commonly associated with vocal cord polyps. Eighty-five percent of direct laryngoscopies were done under general anesthesia with two-thirds utilizing direct suspension microlaryngoscopy. (Auth. Abs. Mod.)

84–1725. Pasurka, S.; Filler, D.; Effenhauser, P. **Lungenkarzinome der Nichtraucher. [Lung Cancer in Non-Smokers.]** *Praxis und Klinik der Pneumologie* 37(1): 1012-1013, October 1983, German.

During the past 6 years, 229 persons (213 men, 16 women) were operated on for lung cancer. Of these, 93 percent (n = 214) were smokers and 7 percent (5 women, 10 men) were nonsmokers. Five of the fifteen nonsmoking patients proved inoperable because of the extent of the tumor and distant metastases. Lung cancer in nonsmokers differed from that in smokers. More women were affected and disease course was asymptomatic without loss of weight. The cancer was often located centrally, thus preventing radical removal, and frequently of the small cell or adenocarcinoma type. (Auth. Abs.)

84–1726. Public Health Reports. **HHS Launches New Cancer Prevention Campaign.** *Public Health Reports* 99(3): 289, May-June 1984.

In the new Cancer Prevention Awareness Program, which hopes to save 95,000 lives/year by the year 2000, the following advice against smoking tops the list of lifestyle factors that can be modified to reduce the risk of cancer: "Don't smoke or use tobacco in any form." The program will emphasize the role of lifestyle factors (smoking, diet, alcohol, occupational exposures, excess sunlight) and environmental factors such as viruses, pollution, and food additives. The National Cancer Institute found (in a national survey) that perceptions have not kept pace with progress; 46 percent of respondents felt there is not much anyone can do to prevent cancer. A general information campaign will be launched, and specific cancer prevention education projects will be developed for audiences at greater-than-average risk. A new booklet, containing anti-smoking advice and other specific actions that can be taken to reduce cancer risk, also answers the most common questions about cancer.

84–1727. Rosenwaike, I. **Changing Patterns of Lung Cancer Among Socio-Cultural Groups in New York City.** *American Journal of Public Health* 74(8): 839-840, August 1984.

The relatively low level of lung cancer mortality among Jewish men, found in an examination of mortality statistics for New York City (1949-1981), is consistent with smoking habits reported for sociocultural groups (although information on patterns for New York City is not available). Data indicate that although lung cancer mortality as a percentage of total mortality increased among both males and females from all three sociocultural groups (Catholics, Protestants, and Jews), the greatest relative increase was among Catholic males. Protestant males showed lower relative increases, and Jews showed markedly lower increases. This pattern was not replicated among women, and in many age groups, Jewish women showed significantly larger increases in mortality relative to Catholic and Protestant women. The higher mortality rates among Jewish women, despite greater educational attainment, suggests that factors other than education are contributing to the findings about smoking habits. The association between smoking and social class is different among men and women in the United States. The persistence of major subgroup differences within specific cohorts, however, would seem to demonstrate some homogeneity of experience that is meaningful in terms of cancer mortality.

84–1728. Salem, G.; Juhl, R.; Schiodt, T. **Oral Malignant and Premalignant Changes in "Shammah"-Users From the Gizan Region, Saudi Arabia.** *Acta Odontologica Scandinavica* 42(1): 41-45, February 1984.

Mucosal lesions compatible with oral leukoplakia were found in approximately 68 percent of the users of "shammah" (native snuff) among the 651 subjects studied. The snuff is prepared by mixing powdered tobacco leaves with hydrated sodium carbonates in various proportions. Lesions occurred more frequently in older age groups, and were almost always at the site where shammah was habitually held. Mucosal lesions were even found in young people, but in shammah users only. Histological examination of biopsy specimens obtained from 29 individuals revealed squamous cell carcinoma in seven cases and carcinoma in situ in two cases, whereas the remaining specimens showed ortho- and para-hyperkeratosis, some with premalignant changes also. Results suggest a possible causal relationship between use of shammah and development of oral premalignant and malignant lesions. (Auth. Abs. Mod.)

84–1729. Silverman, S., Jr.; Gorsky, M.; Lozada, F. **Oral Leukoplakia and Malignant Transformation. A Follow-Up**

Study of 257 Patients. *Cancer* 53(3): 563-568, February 1, 1984.

Among 257 patients with oral leukoplakia, 73 percent used tobacco, and an average 7.2-year followup revealed that 21 of the 45 patients who developed cancer were tobacco users (cigarettes and/or pipe, cigar, or snuff). After diagnosis, 27 percent discontinued tobacco use. At end of followup, 12 percent of ex-smokers, 16 percent of continuing smokers, and 24 percent of nonsmokers had leukoplakia that transformed to carcinoma. It is noted that eight of the malignant transformations were in patients who originally had epithelial dysplasia. Thus risks for malignant transformation included nonsmoking patients, the clinical presence of erythroplasia (erythroleukoplakia), and a clinical verrucous-papillary hyperkeratotic pattern. Duration of leukoplakia progressively increased the total number of malignant transformations, with the highest rate occurring in the second year. Data confirm that oral leukoplakia is a precancerous lesion and that certain characteristics indicate greater risks and warrant consideration of more aggressive management. (Auth. Abs. Mod.)

84–1730. Snowdon, D. A.; Phillips, R. L. **Coffee Consumption and Risk of Fatal Cancers.** *American Journal of Public Health* 74(8): 820-823, August 1984.

The estimated risk of fatal bladder cancer was 30 percent higher in smokers than in those who had never smoked in an analysis of the relationship between coffee consumption and cancer mortality. In 1960, coffee consumption habits, smoking habits, and other lifestyle characteristics of 23,912 white Seventh-day Adventists were assessed by questionnaire. Between 1960 and 1980, deaths due to cancer were identified and a positive association between coffee consumption and fatal colon and bladder cancer was found. The group consuming two or more cups of coffee/day had an estimated relative risk (RR) of 1.7 for fatal colon cancer and 2.0 for fatal bladder cancer compared with the group that consumed less than one cup/day (RR = 1.0). Positive associations were apparently not confounded by age, sex, smoking, or meat consumption habits. No significant or suggestive associations between coffee consumption and fatal pancreatic, breast, and ovarian cancer or a combined group of all other cancer sites were found. (Auth. Abs. Mod.)

84–1731. Spiro, S. G. **Lung Cancer—Areas of Progress.** *Postgraduate Medical Journal* 60: 218-224, March 1984.

In a review of the progress being made in the prevention, detection, and treatment of lung cancer, it is noted that since the main factor controlling incidence is smoking, future trends will depend on what happens to smoking habits in the next decade. Antismoking campaigns have led to significant changes in consumption in many European countries, the United States, and Australia, but in the United Kingdom only the basic minimum is being done, and funding for health education represents only a tiny percentage of the income of tobacco processors. The decline in mortality varies, however, even when smoking habits are taken into account, suggesting that other geographical, occupational, and environmental factors may be involved. Early detection by routine population screening has been less successful than for some other cancers, and alternative diagnostic procedures are discussed, including mediastinoscopy and computed tomography scanning. Results of treatment of non-small-cell lung cancer by surgery, adjuvant therapy, radiotherapy, cytotoxic chemotherapy, and laser therapy are reviewed, noting that while radiotherapy has some palliative value, neither cytotoxic chemotherapy nor laser treatment is likely to be useful in improving survival. Cell biology, histology, and treatment of small cell bronchial cancer are also discussed.

84–1732. Stich, H. F.; Stich, W.; Parida, B. B. **Elevated Frequency of Micronucleated Cells in the Buccal Mucosa of Individuals at High Risk for Oral Cancer: Betel Quid Chewers.** *Cancer Letters* 17(2): 125-134, November-December 1982.

The micronucleus test was applied to buccal mucosa cells from two population groups at high risk for oral cancer: Khasis of the northeastern hill region of India (who eat raw betel nuts together with betel leaves and lime) and residents of the State of Orissa (who chew betel quids consisting mainly of perfumed tobacco, dried betel nut, betel leaf, lime, and several spices). Mironuclei were scored on Feulgen/fast green-stained smear preparations of exfoliated cells obtained by scraping the surface of the buccal mucosa. All 17 raw betel nut eaters and all 20 chewers of betel quids had significantly elevated frequencies of micronucleated mucosa cells over nonchewing controls of comparable ethnic background and dietary habits. The frequencies of micronucleated exfoliated cells were higher at the site within the oral cavity where the quid was kept compared with those at the opposite buccal wall. Micronuclei frequency was lower among individuals chewing a raw betel nut, betel leaf, and lime mixture compared with those using tobacco-, betel nut-, lime-, and betel leaf-containing quids. Micronuclei frequencies in exfoliated human cells seem to represent a useful "internal dosimeter" for estimating exposure to genotoxic and, by implication, carcinogenic agents in the tissue from which cancers will develop. (Auth. Abs.)

84–1733. Trump, B. F.; McDowell, E. M.; Harris, C. C. **Chemical Carcinogenesis in the Tracheobronchial Epithelium.** *Environmental Health Perspectives* 55: 77-84, April 1984.

A research review of pulmonary carcinogenesis notes that cigarette smoking is believed to be the major etiologic factor in bronchogenic carcinoma. Cigarette smoke contains initiators of carcinogenesis, but also many probable promoters and cocarcinogens. It is hypothesized that cigarette smoke may both initiate bronchial cells and promote carcinogenesis in cells that have previously been initiated by smoke or other factors. It is further hypothesized that the mucous cell is the major target for initiation and subsequent tumorigenesis. Comparisons of neoplastic and preneoplastic lesions in human and hamster trachea indicate that bronchogenic carcinomas are typically complex mixtures of three basic phenotypes, the epidermoid, the mucous, and the dense-core granulated (endocrine). Pure forms of these phenotypes are rare, as different cells and even individual cells in single tumors express more than one phenotype. The clinical significance of such phenotypic variability is not yet known, but it is suggested that the ultimate phenotype(s) displayed by the tumor result from the effects of microenvironmental factors upon the initiated cell and its progeny. Bronchial cell types that retain the capacity to divide include the mucous cell, the basal cell, and perhaps the dense-core granulated cell. Studies of epithelial regeneration and preneoplastic lesions suggest that the mucous cell may be pivotal both in the response to injury and in carcinogenesis. (Auth. Abs. Mod.)

84–1734. World Health. **Self-Induced and Avoidable: Oral Cancer in S.-E. Asia.** *World Health* p. 30, May 1984.

Concluding that 90 percent of the more than 100,000 new oral cancers/year in Southeast Asia are caused by tobacco chewing and smoking, participants at a recent World Health Organization meeting recommended that primary prevention be implemented at national levels, with legislative action taken to restrict production, importation, sales, and use of tobacco products. The 5-day meeting, attended by 27 specialists from 12 countries, noted that oral cancer rates are highest among tobacco chewers, the neoplasms almost always occur at the site where the quid is held, and risk rises dramatically for those who keep the quid in the mouth overnight. Citing the 1,000-year-old tobacco chewing habit in India that has been changed through health education, specialists concluded that preventive measures have an impact many times greater than comparable investments in anticancer therapy.

84–1735. Xu, R. H. **Tien-tsin shu Fei-gnhai eu Shien Kwensi-teu Pin-li Tueitsao Enchu. [Case-Controlled Study of the Relationship Between Smoking and Lung Cancer in Tianjin.]** *Chung-Hua Liu Hsing Ping Hsueh Tsa Chih (Chinese Journal of Epidemiology)* 4(4): 193-197, 1983, Chinese.

A significant dose—response relationship between cigarette consumption and relative risk (RR) of lung cancer was found for both sexes in a 1981 retrospective case—control study in six large hospitals in Tianjin, China. Cases (n = 135, 99 males and 36 females) and age-, sex-, race-, and residence-matched controls (n = 135) were all inpatients; 90 percent of cases were diagnosed pathologically. The RR was 5 for male smokers and 4 for female smokers. Mean latency period from smoking onset to detection of lung cancer was 30 years, and displayed a normal distribution curve. (Auth. Abs. Mod.)

See also, **84–1678, 84–1689, 84–1763, 84–1855**

NON—NEOPLASTIC RESPIRATORY DISEASES

84–1736. Adams, L.; Lonsdale, D.; Robinson, M.; Rawbone, R.; Guz, A. **Respiratory Impairment Induced by Smoking in Children in Secondary Schools.** *British Medical Journal* 288(6421): 891-894, March 24, 1984.

Data from a longitudinal study (1975 to 1979) of 405 schoolchildren (average age first year, 13) show that the health of children may be impaired by regular smoking. Respiratory symptoms were more prevalent even after only 1 year of regular smoking and continued to be reported more often while smoking persisted. Subjects underwent a series of respiratory function tests at yearly intervals to monitor lung development. A self-administered questionnaire provided details on smoking habits and respiratory symptoms. Prevalence of smoking increased with age; 4.4 percent of boys and 2.5 percent of girls were regular smokers at age 13. By age 16, 25.2 percent of boys and 27.3 percent of girls reported smoking regularly. Most of those smoking at age 16 had been smoking, at least experimentally, at age 13. Initiation of smoking was clearly associated with the early onset of cough, phlegm production, and shortness of breath on exertion. After 2 years of smoking more than a few cigarettes/day, children who smoked appeared considerably less healthy than nonsmoking peers and showed some evidence of early airway obstruction. (Auth. Abs. Mod.)

84–1737. Bake, B. **Are There Tests of "Small Airways" Dysfunction?** *European Journal of Respiratory Diseases* 65(Supplement 136): 55-58, 1984.

Smokers have been the test group for new means of early detection of small airways dysfunction because chronic obstructive disease and chronic bronchitis develop predominantly in smokers and postmortem studies of young smokers' lungs show consistent pathological changes in the bronchioles. Using a variety of tests, some investigators have claimed that they can detect abnormalities in the small airways. However, the functional consequences of the inflammatory bronchiolitis observed in the lungs of young smokers are not known, and in older smokers, conventional spirometry is abnormal in about 20 percent, indicating large airways involvement. Thus, because there is no definable group with isolated small airways obstruction, what is actually measured by such tests cannot be directly investigated. It is concluded that measures of small airways abnormality are more sensitive to large airways obstruction than conventional tests, but do not specify obstructive disease located in the small airways. A review of some specific tests of lung function is included.

84–1738. Bowden, D. H. **The Alveolar Macrophage.** *Environmental Health Perspectives* 55: 327-341, April 1984.

The pulmonary macrophagic system is critical to the defense of the lung. Under basal conditions, alveolar macrophages are derived from bone marrow. A population of macrophage precursors within the pulmonary interstitium provides a reserve pool capable of proliferation and delivery of phagocytes in response to unusually heavy loads of inhaled particles. As a secretor of proteolytic enzymes, the pulmonary macrophage has been implicated in the pathogenesis of emphysema. Enzymatic secretion usually follows some form of extrinsic stimulus, and since cigarette smoking is a dominant factor in the etiology of emphysema, the response of the macrophage to tobacco smoke is of interest. Macrophages of smokers are different from those of nonsmokers. Smokers have up to four times as many macrophages as nonsmokers, and cells are larger, glucose utilization is three times higher than in controls, and cells exhibit distinctive morphologic features. These changes represent adaptive responses to the increased load, and thus macrophage production is not unusual and is reversible. However, it is suggested that the long-term cost of adaptation may compromise the defenses of the host. Although the protective role is dominant, macrophages may participate, directly or indirectly, in the genesis of interstitial fibrosis and emphysema. Such inappropriate responses involve interactions with fibroblastic cells and tissue injury initiated by proteases secreted by the macrophage. (Auth. Abs. Mod.)

84–1739. Buist, A. S. **Current Status of Small Airways Disease.** *Chest* 86(1): 100-105, July 1984.

There is convincing evidence that although inflammatory changes in the small airways are common in cigarette smokers, such changes do not necessarily lead to emphysema, which is usually the cause of chronic airflow obstruction. Tests of small airways function appear to be quite sensitive for detecting early airflow obstruction that is probably synonymous with inflammatory disease in the small airways. However, the relatively high sensitivity of such tests is not matched by a high specificity or a high predictive value in identifying individuals who will progress from mild airflow obstruction to clinical airflow obstruction. This is probably because inflammatory disease in the small airways, although associated with emphysema in that both conditions are found in cigarette smokers, is not causally related to emphysema. Tests of small airways function still have an important place in epidemiologic studies and in other research studies. Clinically, measurements obtained from the spirogram and from the maximal expiratory flow-volume (MEFV) curve are useful in identifying the individual with mild airflow obstruction. The single breath N_2 test and the MEFV curve while breathing gases of different densities are still largely research tools. (Auth. Abs. Mod.)

84–1740. Burrows, B. **Possible Pathogenetic Mechanisms in Chronic Airflow Obstruction.** *Chest* 85(6, Supplement): 13S-15S, June 1984.

Elevated serum immunoglobulin E (IgE) in smokers does not appear to be specific for common seasonal aeroallergens. A type of IgE that is not aeroallergen specific (such as that induced by smoking) is associated with a low percent predicted forced expiratory volume in 1 sec (FEV_1), but only when there are symptoms suggesting an "asthmatic bronchitic" type of disease. Current hypotheses of the pathogenesis of airways obstructive diseases are based on findings that suggest some of the adverse effects of smoking may be immunologically mediated, leading to chronic asthmatic bronchitis. An asthmatic predisposition may be essential in developing the syndrome, which would be compatible with the so-called Dutch theory of the development of airways obstruction. A more insidious development of ventilatory impairment, not accompanied by bronchitis or asthma complaints, probably results from obliterative disease of small airways and emphysema (conforming to the so-called British hypothesis). The difference between the Dutch and British hypotheses may be a reflection of different types of disease present in London transport workers compared with residents of small communities in the Netherlands in the 1950s and 1960s. When looking at the pathogenesis of airways obstruction 25 years later, it may be unwise to lump all FEV_1 abnormalities under the rubric of chronic obstructive pulmonary disease.

84–1741. Carrell, R. W. **α_1-Antitrypsin, Emphysema and Smoking.** *New Zealand Medical Journal* 97(756): 327-328, May 23, 1984.

In persons with genetic α_1-antitrypsin deficiency, onset of emphysema and death can occur 20 years earlier in smokers than in nonsmokers. The observation that emphysema was a consequence of a protease—antiprotease imbalance provided an explanation of smoking's etiological role: chronic smoke inhalation results in up to a 10-fold rise in pulmonary alveolar macrophages, depending on the number of cigarettes consumed. Macrophages also change, increasing proteolytic activity and secreting a chemotactic attractant to bring leukocytes to the lungs. Tobacco smoke stimulates leukocytes to secrete proteolytic enzymes (primarily elastase), and oxidants, which are responsible for inactivation of already low levels of α_1-antitrypsin. Thus emphysema results from chronic smoke exposure or inflammation, owing to attacks from leukocytic enzymes and exacerbated by loss or inactivation of protective α_1-antitrypsin. Biochemical interactions involved in the destruction of lung tissue are reviewed.

84–1742. Catz, E. G.; Speir, W. A., Jr. **α_1-Antitrypsin Deficiency.** *Southern Medical Journal* 77(4): 479-483, April 1984.

Data suggest that smoking may increase elastase release via several mechanisms while suppressing α_1-antitrypsin (α_1-AT) activity against elastase, thus enhancing the effects of α_1-AT deficiency. The effects of smoking are included in a review of the pathophysiology, clinical course, and extrapulmonary manifestations of α_1-AT deficiency. The deficiency is inherited, with multiple codominant alleles at a single autosomal locus. Homozygous individuals, with severely reduced α_1-AT levels, have dyspnea, pulmonary function abnormalities, and respiratory disability from emphysema, usually in the fifth decade of life. The clinical course in smokers is more rapidly and consistently progressive, with onset of symptoms, abnormalities of lung function, and age at death from respiratory failure occurring at least one decade earlier than in nonsmokers. Heterozygous individuals have intermediate α_1-AT levels and a more benign clinical course. Heterozygous smokers may have mild pulmonary function abnormalities, but these are of uncertain clinical significance. Hepatic involvement with transient neonatal hepatitis and cirrhosis with subsequent liver failure in adulthood represent the major extrapulmonary manifestations, occurring in 10 percent of homozygous individuals. (Auth. Abs. Mod.)

84–1743. Celli, B. R.; Rodriguez, K. S.; Snider, G. L. **A Controlled Trial of Intermittent Positive Pressure Breathing, Incentive Spirometry, and Deep Breathing Exercises in Preventing Pulmonary Complications After Abdominal Surgery.** *American Review of Respiratory Disease* 130(1): 12-15, July 1984.

It is suggested that the lack of an association between smoking habits and complications following surgery was because only 31 of the 172 patients were smokers with a history of >15 pack-years. Subjects were randomized into (1) controls (n=44) who received no treatment; (2) 45 patients that received intermittent positive pressure breathing therapy (IPPB) for 15 minutes, four times/day; (3) 42 who were treated with incentive spirometry (IS) four times/day; and (4) 41 who carried out deep breathing exercises (DBE) for 15 minutes, four times/day. The frequency of development of pulmonary complications was 48 percent in controls, 22 percent in the IPPB group ($p<0.05$), 21 percent in the IS group ($p<0.05$), and 22 percent in the DBE group ($p<0.05$). Side effects of respiratory treatment were observed only in the IPPB group (18 percent; $p<0.05$). Hospital stay in patients undergoing upper abdominal surgery was significantly shorter in the IS group than in controls, a difference not observed for other treatment groups. It is concluded that IPPB, IS, and DBE were equally effective in preventing pulmonary complications after abdominal surgery, although IS may be the treatment of choice in upper abdominal procedures. (Auth. Abs. Mod.)

84–1744. Chaieb, J. A.; Vitola, D.; Silva, M. S.; Clausell, N.; Neff, F. S.; Levin, H. **Epidemiologia das Doencas Respiratorias Obstrutivas em Relacao com o Habito de Fumar. [Epidemiology of Obstructive Respiratory Diseases and Smoking.]** *Boletin de la Oficina Sanitaria Panamericana* 96(2): 119-133, February 1984, Portuguese.

Significant differences in respiratory flow rates between smokers, ex-smokers, and nonsmokers were found in a population sample (n=540) from Porto Alegre, Brazil. Average respiratory flow found in different age groups of the 316 smokers, the 102 ex-smokers, and the control group of 122 nonsmokers were compared with predicted flows. Flow reduction in nonsmokers were as predicted. In males over age 55 and females between 45 and 64 years of age, a significantly greater reduction in flow was observed in smokers than in nonsmokers. The average age at which individuals in the group stopped smoking was 55±12 years, suggesting that people give up smoking chiefly because of the onset of serious respiratory symptoms. It also indicates that the age of ±55 years should be taken as an indicator in assessing the effects of campaigns against smoking. (Auth. Abs. Mod.)

84–1745. Chow, C. K.; Chen, L. H.; Thacker, R. R.; Griffith, R. B. **Dietary Vitamin E and Pulmonary Biochemical Responses of Rats to Cigarette Smoking.** *Environmental Research* 34(1): 8-17, June 1984.

Results suggest that the nutritional status of vitamin E may influence the cellular susceptibility of rats to cigarette smoke. Young male rats maintained on a basal vitamin E-deficient diet with/without 100 ppm vitamin E supplementation for 4 or 5 weeks were exposed to either sham or cigarette smoke for up to 7 days. Higher mortality was observed in animals fed the vitamin E-deficient diet than in the supplemented group when subjected to acute levels of cigarette smoke. Relative to the respective sham groups, a greater alteration of biochemical parameters, such as reduced glutathione (GSH) and related enzymes, was found in the lungs of smoke-exposed rats fed the deficient diet than in the supplemented group. Animal lungs exhibited a greater biochemical response to whole smoke than to the gaseous phase of smoke. (Auth. Abs. Mod.)

84–1746. Churg, A.; Wright, J. L. **Small Airways Disease Induced by Asbestos and Nonasbestos Mineral Dusts.** *Chest* 85(6, Supplement): 36S-38S, June 1984.

Results show that in workers exposed to a variety of mineral dusts (including asbestos) similar lesions of small airways are found that differ from those seen in pure cigarette smokers; however, widespread fibrosis of the membranous bronchioles was also found. These lesions were not morphologically different from those of cigarette smokers. Subjects were 15 asbestos-exposed and 7 nonasbestos dust-exposed workers, matched by age, sex, and smoking history with controls who had no occupational dust exposure. Grading analysis showed that the similar lesions were more common and more severe in subjects than changes seen in matched cigarette-smoking controls. Whether such changes are additive or multiplicative effects of the combination of cigarette smoke and mineral dust is an important and unanswered question. The changes are important because pathologic alterations in membranous bronchioles have been clearly correlated with airflow obstruction in cigarette smokers, and the locus of obstructive effects from mineral dusts may also be at the level of the membranous bronchiole.

84–1747. Cosio, M. G.; Angus, E.; Shiner, R.; Wang, N. S.; Ghezzo, H.; Saetta, M.; Esau, S. **Scanning Electron Microscopy (SEM) of the Lung in Smokers; Its Relationship to Function.** *Chest* 85(6, Supplement): 35S, June 1984.

Scanning electron microscopy (SEM) was used to determine whether "fenestrae" (holes) might be the first step in the production of smokers' centrilobular emphysema. In 12 patients undergoing surgery, five template sections were taken from a subpleural slice of lung and adjacent sections processed for SEM and light microscopy. Results indicate that fenestrae increase in size and decrease in number with increasing disease, suggesting coalescence of holes. The changes probably represent the earliest morphologic emphysema and may be responsible for functional abnormalities.

84–1748. Crystal, R. G.; Bitterman, P. B.; Rennard, S. I.; Hance, A. J.; Keogh, B. A. **Interstitial Lung Diseases of Unknown Cause. Disorders Characterized by Chronic Inflammation of the Lower Respiratory Tract. (Second of Two Parts).** *New England Journal of Medicine* 310(4): 235-244, January 26, 1984.

In an evaluation of alveolitis associated with interstitial lung diseases of unknown cause, it is important to recognize that cigarette smoking causes an alveolitis characterized by a threefold to fivefold increase in the number of alveolar macrophages and a small increase in the number of neutrophils. From a clinical viewpoint, lavage analyses in smokers must be evaluated with the knowledge that inflammation induced by cigarette smoking probably influences the distribution of the effector cells recovered. The general concepts of pathogenesis; role of lymphocytes, alveolar macrophages, and other effector cells; role of immune complexes and complement; and anergy and immune functions in skin and blood associated with sarcoidosis are described. In discussing histiocytosis X, it is noted that almost all patients are either current or former smokers, suggesting that components of cigarette smoke may play a part in pathogenesis, involving abnormal responses by cells of the mononuclear phagocyte system. General concepts of diagnosis, staging, and treatment of idiopathic pulmonary fibrosis, sarcoidosis, and histiocytosis X are also included.

84–1749. Decramer, M.; Demedts, M.; Van de Woestijne, K. P. **Isocapnic Hyperventilation With Cold Air in Healthy Nonsmokers, Smokers and Asthmatic Subjects.** *Clinical Respiratory Physiology* 20(3): 237-243, 1984.

Isocapnic hyperventilation with subfreezing air was performed twice by 15 healthy nonsmokers, 10 asymptomatic smokers, and 9 asthmatics. All subjects had normal ventilatory function and airway resistance (Raw) before challenge. In the first session, total respiratory resistance (Rrs) and reactance were measured at various frequencies, using a forced oscillation technique. In the second session, vital capacity (VC), forced expiratory volume in 1 sec (FEV_1), maximal expiratory flow rates (FEF), and Raw were determined. In nonsmokers, no changes in FEV_1, FEF, or Raw were observed, whereas Rrs increased significantly without change in resonant frequency or in Rrs—frequency relationship, suggesting constriction of central airways only. In smokers, Rrs showed a similar, though longer lasting, increase. A significant change of the Rrs—frequency relationship and reduction in FEF at 50 percent of VC was also found, suggesting peripheral airway involvement. In asthmatics, bronchial reactivity was more pronounced, resulting in significant changes in all parameters, compatible with more generalized constriction of the peripheral and central airways. (Auth. Abs. Mod.)

84–1750. Duncan, D. B.; Greaney, P. P. **Passive Smoking and Uptake of Carbon Monoxide in Flight Attendants. (Letter).** *Journal of the American Medical Association* 251(20): 2658-2659, May 25, 1984.

Exposure to passive smoke during flights did not significantly alter carbon monoxide (CO) levels in expired air of commercial airline attendants. Sixteen nonsmoking women aged 35 to 48 years, four of whom worked in nonsmoking sections, completed health history questionnaires. After 10 hours of flight time (with 1 hour stopover), expired air (end tidal) CO levels were measured. No increase in expired CO concentration was observed, and when those who worked in nonsmoking areas were excluded from analysis, results were not substantially altered.

84–1751. Gadek, J. E.; Fells, G. A.; Zimmerman, R. L.; Crystal, R. G. **Role of Connective Tissue Proteases in the Pathogenesis of Chronic Inflammatory Lung Disease.** *Environmental Health Perspectives* 55: 297-306, April 1984.

A review of the role of proteolysis in human lung disease suggests that cigarette smoke adversely affects both sides of the protease—antiprotease balance. The oxidative elements of smoke reduce the capacity of α_1-antitrypsin to inhibit elastase, while smoke provokes an influx of neutrophils and the resulting increase in elastase activity. Also, a smoker's lung has little anticollagenase and 40 to 50 percent antielastase. The net effect is continuous expression of both collagenase and elastase, since there are insufficient amounts of inhibitors. Normal homeostatic mechanisms, which both regulate the influx of protease-producing cells to the lung parenchyma and modulate protease activity via intermolecular complexes, are explored. Also described are the connective tissue matrix of human alveoli and the role of individual components such as collagen, elastic fibers, proteoglycans, and other glycoproteins. Connective tissue proteases relevant to diseases of the alveolar structure (collagenase, elastase, and other proteases) are discussed.

84–1752. Gardner, D. E. **Alterations in Macrophage Functions by Environmental Chemicals.** *Environmental Health Perspectives* 55: 343-358, April 1984.

An overview of the effects of tobacco smoke and other environmental agents on the biochemical, physiological, and immunological functioning of pulmonary alveolar macrophages is presented. Specifically discussed are the morphologic, phagocytic, and biochemical changes induced by cigarette smoking. In addition to filling the cytoplasm with pigmented residues, smoke causes an increase in mean maximum diameter. Smokers' macrophages are more rounded, with little or no ruffling of the membrane, and show changes in the volume density of the mitochondria, surface density of the rough endoplasmic reticulum, and concentration of cytoplasmic lipids. Cigarette smoke also variably influences chemotaxis and has a cumulative effect on phagocytic functioning. Smokers' macrophages show a twofold increase in oxygen consumption and hydrogen peroxide release, and higher aryl hydrocarbon hydroxylase activity. Other alterations in cell types, numbers, and viability are also explored. The effects of environmental agents on macrophage defenses against bacterial and viral attack and the influence of the surrounding respiratory milieu are also covered.

84–1753. Gardner, G.; Frank, A. L.; Taber, L. H. **Effects of Social and Family Factors on Viral Respiratory Infection and Illness in the First Year of Life.** *Journal of Epidemiology and Community Health* 38(1): 42-48, March 1984.

The only significant difference in viral infection or respiratory illness seen with parental smoking as an isolated factor was the relationship between pneumonia and parental smoking (especially mothers at home who smoked). A total of 131 infants were monitored from birth through the first year of life for respiratory viral infection and illness and evaluated for a relationship with social and familial factors. Results showed no general patterns of association between viral infection and study factors, but there were several significant individual associations. Excess influenza virus infection was found for black infants, infants with at least one sibling, and especially those with school-age siblings. Rhinovirus infection rates were highest among girls attending day care. In addition, significantly higher rates of lower respiratory disease (LRD) were seen in day care infants and low socioeconomic infants, and a definite trend to increasing amounts of LRD was seen with increasing family size. Protection from LRD seen in girls was apparently lost in day care. (Auth. Abs. Mod.)

84–1754. Glazier, A. J.; Hunninghake, G. W. **Alveolar Macrophages and Lung Connective Tissue.** *Current Problems in Clinical Biochemistry* 13: 202-211, 1983.

Pulmonary emphysema occurs in two groups: people with inherited α_1-antitrypsin deficiency and cigarette smokers. Particulates in tobacco smoke oxidize a methionine residue of the α_1-antitrypsin molecule, inhibiting its capacity to inactivate neutrophil elastase. Thus smoking results in a functional equivalent of α_1-antitrypsin deficiency. Cigarette smoke also stimulates alveolar macrophages to release a chemotactic factor for neutrophils, which stimulates neutrophils to release elastase. Since this elastase is not adequately inactivated by smoke-oxidized α_1-antitrypsin, the result is destruction of the elastin proteins in the lung. Interstitial lung disease is also discussed, with respect to the modulation of collagen production, disordering and derangement of collagen, and the interaction of alveolar macrophages with lung parenchymal cells.

84–1755. Groth, S.; Lindell, S. E.; Kabiraj, M. U.; Bulow, K.; Arborelius, M., Jr.; Simonsson, B. G. **Bronchial Reactivity and Small Airway Dysfunction in Subjects With Intermediate Alpha$_1$-Antitrypsin Deficiency.** *Clinical Respiratory Physiology* 20(3): 279-284, 1984.

Results indicate that although no direct correlation between small airways and bronchial reactivity was found, smoking correlated with both airway dysfunction and increased reactivity, suggesting a causal relationship between small airways dysfunction and bronchial reactivity. Bronchial reactivity to methacholine (MCh), spirometry, and several variables thought to be sensitive to small airways function were studied to assess a possible relationship between bronchial reactivity and signs of small airways dysfunction. There were significantly more smokers ($n=14$) than ex-smokers ($n=7$) or nonsmokers ($n=2$) among the 23 subjects who were found to have increased bronchial reactivity. Smokers who had increased reactivity to MCh challenge had significantly higher percent of closing capacity, residual volume/total lung capacity, and volume of trapped gas than smokers without increased reactivity. This difference was not seen for percent closing volume, slope index, washout volume, or ΔN_2 percent/l. There was no difference between PiM and PiMZ subjects on any lung function variables or response to MCh. (Auth. Abs. Mod.)

84–1756. Hayden, S. P.; Pincock, A. C.; Hayden, J.; Tyler, L. E.; Cross, K. W.; Bishop, J. M. **Respiratory Symptoms and Pulmonary Function of Welders in the Engineering Industry.** *Thorax* 39(6): 442-447, June 1984.

Respiratory symptoms, smoking habits, chest radiographs, sickness absence, and pulmonary function were studied in 258 welders and an equal number of matched controls in three engineering factories. Welders who smoked had a higher frequency of chronic phlegm production than control subjects, but there was no difference in cough or dyspnea. The frequency of abnormality on chest radiographs was low and similar in welders and controls. Upper respiratory infections were a more frequent cause of sickness absence in welders than in controls, but no difference was found in other respiratory diseases. Smokers, however, had nearly twice as many sickness absence days as nonsmokers in both groups. Forced expiratory volume in 1 sec and peak expiratory flow rate were similar in welders and controls, but smokers showed significantly lower values than nonsmokers. In a subset of 186 subjects, maximum expiratory flow rate at low lung volumes was significantly less in welders who smoked than in control subjects who smoked, but there was no difference in nonsmokers. Welders in the engineering industry appear to have no increased risk of chronic obstructive lung disease. (Auth. Abs. Mod.)

84–1757. Higgins, M. **Epidemiology of COPD: State of the Art.** *Chest* 85(6, Supplement): 3S-8S, June 1984.

In a review of the epidemiology of chronic obstructive pulmonary disease (COPD), cigarette smoking is discussed as a major risk factor. Data from longitudinal, cross-sectional, and case—control studies show that smokers have higher death rates for chronic bronchitis and emphysema; higher prevalence and incidence rates for chronic bronchitis, emphysema, and obstructive airways disease; higher frequencies of respiratory symptoms and lung function abnormalities; and a greater average annual rate of decline in forced expiratory volume than nonsmokers. Differences increase with increasing cigarette consumption. Ex-smokers have fewer symptoms than continuing smokers, but lost function is not regained. Prognosis is less hopeful if cessation occurs at older ages, among heavy smokers, or among those with advanced disease. Factors influencing the onset of COPD in smokers include interactions with other environmental hazards, tar and nicotine intake, and passive smoking. Other subjects discussed include reduced lung function, occupational exposures, air pollution, familial and host factors, respiratory tract infections, and risk estimates.

84–1758. Hudson, L. D. **Management of COPD. State of the Art.** *Chest* 85(6, Supplement): 76S-81S, June 1984.

Because smoking has been identified as the major cause of chronic obstructive pulmonary disease (COPD), prevention of smoking, or cessation early in the course of the disease, is the best approach to management. Although the evidence that quitting slows the rate of obstruction to that of nonsmokers has been criticized, it is nonetheless reasonable to operate on the premise that cessation will slow progression of the disease, and patients who smoke should be advised to quit. It is noted that surveys of ex-smokers indicate physician's advice was a major factor in the decision to stop smoking. For certain patients, nicotine chewing gum (now available by prescription) may be an aid for quitting. Also discussed are bronchial reactivity, infections, home oxygen therapy, use of drugs affecting ventilatory drive, physical measures, and psychosocial considerations in the treatment of COPD.

84–1759. Kanner, R. E.; Renzetti, A. D., Jr. **Predictors of Spirometric Changes and Mortality in the Obstructive Airway Disorders.** *Chest* 85(6, Supplement): 15S-17S, June 1984.

Current smoking was significantly associated with lower probability of 12-year survival in a longitudinal study of 140 subjects with chronic airflow limitation. Mean age of patients (79 percent males) was 58.4±6.9 years; 52.1 percent were current smokers, 31.4 percent were ex-smokers, and 16.4 percent never smoked. Years of smoking correlated with annual decline in forced vital capacity (FVC) and forced expiratory volume in 1 sec ($p<0.001$). Those diagnosed as having chronic bronchitis with emphysema (CBE, 58 percent of subjects) had a mean of 37.1 years of smoking compared with 24 years in emphysema patients, 25.9 years in chronic bronchitis patients, 5.3 years in asthmatics, and 3.2 years among those with bronchiectasis. Mean annual declines in FVC were 129.7 ml for CBE patients, 90.6 ml for emphysema patients, and 86 ml for asthmatics. In addition to cigarette consumption, α_1-antitrypsin, age, airway reactivity, and lower respiratory tract illness were all significantly associated with annual rate of change in lung function. The 12-year survival probability was 0.86 for never smokers, 0.79 for former smokers, and 0.64 for current smokers. The role of respiratory infections is also discussed.

84–1760. Kilburn, K. H. **Particles Causing Lung Disease.** *Environmental Health Perspectives* 55: 97-109, April 1984.

Cigarette smoke, a complex combination of poorly digestive lipids, complex carbohydrates, and active chemicals, is most often responsible for human lung disease due to particle inhalation. The lung has a limited number of reactions to inhaled particulates, with the resulting disease dependent on the location (conducting airways, terminal bronchioles, and alveoli) and nature of inflammation induced (acute, subacute, or chronic). The most insidious and probably most important lung disease is smoking-related bronchiolar obstruction, producing progressive impairment of airflow. Many different agents cause narrowing of conducting airways (asthma) and some of these cause permanent distortion or obliteration of airways as well. Terminal bronchioles appear to be particularly susceptible to particles that cause goblet cell metaplasia, mucous plugging, and ultimately peribronchiolar fibrosis. In contrast, emphysema (a catabolic response) appears frequently to result from leakage or release of lysosomal proteases into the lung during processing of cigarette smoke particles. Cancer is the last outcome at the bronchial level, and appears to depend on continuous exposure to, or retention of, an agent in the airway and failure of the affected cells to be exfoliated, which may be due to squamous metaplasia. The functioning of the normal healthy lung and mechanisms of particle-induced damage are explored. (Auth. Abs. Mod.)

84–1761. Kimbel, P. **Proteolytic Mechanisms of Lung Injury in the Pathogenesis of Emphysema.** *Chest* 85(6, Supplement): 39S-41S, June 1984.

Current data on the pathogenesis of emphysema suggest the existence of a complex system of interactions between cigarette smoke, phagocytic cells, elastases, protease inhibitors, oxidants, lung connective tissue, and other mediators and inhibitors of inflammation. The importance of elastin degradation in the development of emphysema has been demonstrated, and it is probable that a significant level of elastase is present in the lungs of smokers, which produces an imbalance in the enzyme-inhibitor system, resulting in free elastase when the inhibitor is also functionally impaired by cigarette smoke. The inhibitory capacity of alpha$_1$-antitrypsin may be exceeded by excessive enzyme concentrations. Naturally occurring defenses may be one of the determinants for the development of emphysema, while exposure to pollutants such as ozone may augment lung damage. Although elimination of smoking would probably remove emphysema from the list of major illnesses, it is still not known whether progression of the disease can be halted by any therapeutic agents once it is firmly established.

84–1762. Knudson, R. J.; Bloom, J. W.; Knudson, D. E.; Kaltenborn, W. T. **Subclinical Effects of Smoking. Physiologic Comparison of Healthy Middle-Aged Smokers and Nonsmokers and Interrelationships of Lung Function Measurements.** *Chest* 86(1): 20-29, July 1984.

Although very few of the 22 healthy middle-aged smokers and 19 nonsmokers studied had abnormal lung function, there were significant differences in most indices of function between smokers and nonsmokers. Measurements of ventilatory function, distribution of ventilation, diffusing capacity, and lung mechanics were taken to assess the effects of cigarette smoking and the interrelationships of several indices of lung function. For the total group studied, there were also significant correlations between various indices of function. A significant proportion of the variance in diffusing capacity and in diffusing capacity/liter of lung volume can be accounted for by an index of lung recoil that may, in turn, be related to size of terminal air spaces. (Auth. Abs. Mod.)

84–1763. Kubik, A. **The Influence of Smoking and Other Etiopathogenetic Factors on the Incidence of Bronchogenic Carcinoma and Chronic Nonspecific Respiratory Diseases.** *Czechoslovak Medicine* 7(1): 25-34, 1984.

Longitudinal epidemiological studies carried out in the Kolin District of Czechoslovakia over the past 20 years confirm the etiopathogenetic role of cigarettes in bronchogenic carcinoma and chronic bronchitis. Among those aged ≥ 15 years, the proportion of smokers was 57 percent in males and 14 percent in females. The incidence of bronchogenic carcinoma, verified by histological or cytological examination, was approximately 50 times higher in males from 40 to 64 years of age who had smoked $>200{,}000$ cigarettes than in nonsmokers. In moderate smokers, the incidence was 10 times higher than in nonsmokers. The frequency of new cases of bronchogenic carcinoma was much higher in males with chronic respiratory symptoms and with tuberculous or posttuberculous fibrotic lesions in the lungs. Overall prevalence of chronic bronchitis in 1972 was 13 percent for males and 4 percent for females. Among those who had smoked 100,000 cigarettes, 28 percent of males and 20 percent of females had symptoms suggestive of chronic bronchitis compared with 5 percent of male and 3 percent of female nonsmokers. The incidence of bronchogenic carcinoma and chronic nonspecific respiratory diseases was further increased by the interaction of two or more risk factors. (Auth. Abs. Mod.)

84–1764. Leitch, A. G.; Lumb, E. M.; Kay, A. B. **Disodium Cromoglycate Relieves Symptoms in Symptomatic Young Smokers. A Double Blind Placebo Controlled Trial.** *Allergy* 39(3): 211-215, April 1984.

Administration of disodium cromoglycate (DSCG) produced significant improvement in cough ($p<0.01$) and wheezing ($p<0.025$), but not sputum production, in a double-blind, placebo-controlled study of 32 symptomatic young smokers. Subjects had regular morning cough and sputum with the sensation of wheeze, but were not asthmatics as determined by usual criteria. No significant changes were observed in sputum or serum eosinophils, immunoglobulins E, G, or A, or sputum histamine concentrations after 3 weeks of either DSCG (20 mg) or placebo three times/day. The placebo was not associated with improvements in symptoms of cough and wheezing. It is suggested that these changes are due to an effect of DSCG on the irritant receptor-mediated reflex response to cigarette smoke rather than the involvement of mast cells. (Auth. Abs. Mod.)

84–1765. Luce, J. M. **Clinical Risk Factors for Postoperative Pulmonary Complications.** *Respiratory Care* 29(5): 484-495, May 1984.

An overview of clinical factors indicative of postoperative pulmonary complications notes that complications are most common among cigarette smokers, with an incidence of 14.8 percent compared with 6.3 percent in nonsmoking patients. Smoking also may account for sex-related differences in complication rates. It is speculated that the greater closing volume, respiratory symptoms, and other lung function abnormalities found in smokers predisposes to postoperative atelectasis and hypoxemia. However, incidence of postoperative pulmonary complication is greater in patients with chronic respiratory diseases (26.4 percent) than in smokers without such disorders (8.2 percent). It is concluded that smoking-related lung disease rather than cigarette use per se is the more important indicator of risk. Other factors reviewed include type of anesthesia, type and duration of surgery, age, general physical status, body weight, heart disease, and lung disease. Assessment of clinical risk factors and areas for further investigation are also discussed.

84–1766. Lynn, W. S. **Control of Cellular Influx in Lung and Its Role in Pulmonary Toxicology.** *Environmental Health Perspectives* 55: 307-311, April 1984.

The normal pulmonary influx of cytotoxic inflammatory cells in response to external toxins is now thought to be etiologic in many of the disease syndromes of man, such as bronchitis and emphysema. Many types of effector inflammatory cells are involved, e.g., eosinophils, neutrophils, T-lymphocytes, and monocytes. Inhalation of cigarette smoke, ozone, or elevated concentrations of oxygen or intravenous injection of Freund's adjuvant, silica, or aluminum particles results in accumulation of polymorphonuclear leukocytes acutely and of macrophages subsequently into alveolar and interstitial compartments. Diseases are characterized either by tissue destruction or by tissue hyperplasia. The many agents that initiate the influx and cytotoxic secretions by cells are generally not cell specific. Other agents, such as linoleic acid, digitonin, and hydroxy fatty acids that are not bound by specific receptors also activate motility of inflammatory cells. The precise role of the multiple cytotoxins in specific cellular fluxes in most pulmonary disease remains undefined. Similarly, the mechanism of cytotoxicity used by specific invading cells in specific pulmonary syndromes remains unclear. In general, macrophages are thought to destroy by specific proteases, neutrophils by oxidant radicals, and proteases and eosinophils by basic surface active peptides. T-cells kill by unknown mechanisms. However, in specific clinical syndromes, it is usually not clear which cell is the cytotoxic culprit, nor is the mechanism of destruction usually known. (Auth. Abs. Mod.)

84–1767. Minty, B. D.; Royston, D.; Jones, J. G.; Hulands, G. H. **The Effect of Nicotine on Pulmonary Epithelial Permeability in Man.** *Chest* 86(1): 72-74, July 1984.

Whole smoke, but not nicotine alone, significantly increased the permeability of lung epithelium in five healthy nonsmokers (aged 39 to 53) who agreed to take up smoking (10 cigarettes/day for 3 days) and five volunteers (aged 26 to 36) who chewed 2 mg nicotine gum 12 times/day for 7 days. An index of pulmonary permeability was derived from the rate of clearance from the lung into the blood of ^{99m}Tc diethylenetriamine pentacetate. In the volunteers who chewed nicotine gum, there was no change in permeability for the 7-day period. This lack of effect of nicotine alone was in striking contrast to the increase in epithelial permeability observed in the subjects who smoked. (Auth. Abs. Mod.)

84–1768. Oldham, P. D.; Seaton, A. **Coal and the Lung. (Letters).** *Thorax* 39(5): 397-398, May 1984.

In comments on an editorial regarding the effects of coal dust and smoking on lung function, it is pointed out that (1) the question of direction of bias among nonrespondents is moot (the proportion of nonrespondents in the study (*Thorax* 37(3): 193-197, March 1982) was too large); (2) considering the smoking effect as additive is overly simplistic; and (3) dust exposure estimates were speculative. The author replies that recent studies show that miners leaving the collieries before retirement had slightly more lung function reduction than those who remained and that the significant effect of dust exposure cannot be distinguished by smoking status. New information has provided a rational basis for dust control strategies to protect workers' health. See also 82-1326.

84–1769. Osmanliev, D. P.; Davies, E. E.; Pride, N. B. **Transit Time Analysis of the Forced Expiratory Spirogram in Male Smokers.** *Clinical Respiratory Physiology* 20(3): 285-293, 1984.

Comparison of transit times from 149 young and middle-aged male smokers and 53 nonsmokers showed that there is probably an increase in dispersion of forced transit times in many young smokers when usual spirographic indices are within normal limits. Transit time analysis has been proposed as a simple and sensitive method for detecting slowing and nonuniformity of lung emptying in airway disease. The first moment (α_1, mean transit time) and second moment (α_2) about the origin of the forced expiratory spirogram were measured. Values of α_1 and $\alpha_2^{1/2}$ (calculated from the first 6 seconds of expiration) increased with age in both smokers and nonsmokers and were significantly greater in older smokers than in nonsmokers. The rate of increase in α_1 and $\alpha_2^{1/2}$ was most rapid in smokers between 24 and 36 years. Changes in α_1 were closely related to changes in forced expiratory volume in 1 sec/forced vital capacity (FEV_1/FVC) ratio. Values of $\alpha_2^{1/2}$ were frequently slightly increased in young smokers and in older smokers without definite abnormality of FEV_1/FVC, or α_1, probably because $\alpha_2^{1/2}$ is more strongly influenced by the final part of the spirogram. Problems persist in determining the best method for expressing abnormality in $\alpha_2^{1/2}$, in allowing for premature termination of the spirogram, and for assessing dispersion of transit times. It is concluded that α_1 demonstrates abnormalities not detected by α_1 in many young smokers and in older smokers with no or only slight increase in α_1. Once α_1 is definitely increased, measuring $\alpha_2^{1/2}$ provides little additional information. Results suggest transit time analysis is likely to be a suitable method for detecting minor airway disease in population surveys, but its usefulness in comparison with other available tests remains to be established. (Auth. Abs. Mod.)

84–1770. Pandey, M. R. **Domestic Smoke Pollution and Chronic Bronchitis in a Rural Community of the Hill Region of Nepal.** *Thorax* 39(5): 337-339, May 1984.

Prevalence of chronic bronchitis in rural Nepal was significantly associated with smoking, and for female nonsmokers and ex-smokers of both sexes, with exposure to smoke from heating and cooking. An earlier study of chronic bronchitis in a rural community of the Hill Region of Nepal using Medical Research Council criteria showed a high prevalence rate that was more or less equal in the two sexes. Most of the women are exposed to domestic smoke caused by wood and straw fires used for cooking and heating purposes in ill-ventilated houses without chimneys. Data from 1,375 individuals were analyzed to determine the relationship between domestic smoke pollution and chronic bronchitis. Exposure to domestic smoke was assessed according to the average time/day spent near the fireplace. The association between chronic bronchitis and cigarette smoking was confirmed ($p<0.01$). Age-adjusted prevalence was highest in ex-smokers at all exposure levels (70 percent in men and 63 percent in women at the highest exposure level), suggesting that many had stopped smoking after developing symptoms. In the living conditions existing in rural Nepal, domestic smoke pollution is an important contributing factor in the development of chronic bronchitis. (Auth. Abs. Mod.)

84–1771. Philpot, R. M.; Smith, B. R. **Role of Cytochrome P-450 and Related Enzymes in the Pulmonary Metabolism of Xenobiotics.** *Environmental Health Perspectives* 55: 359-367, April 1984.

In a review of metabolism of xenobiotics in the lung it is noted that most human lung tumors associated with smoking originate from the bronchiolar epithelium, where characterization of polycyclic aromatic hydrocarbons (PAH) metabolism is important. However, study results have shown no clear relationship between pulmonary metabolism of benzo(*a*)pyrene and the susceptibility of lung to PAH-induced carcinogenesis. The lung metabolizes a wide variety of xenobiotics and, in the process, forms products that may be more or less toxic than the parent compound. The consequence of metabolism, activation or detoxication, is a function of the nature of the substrate and of the characteristics and concentrations of the enzymes involved. As a result, the biotransformation of xenobiotics can lead to excretion or to formation of reactive products that produce deleterious effects by binding covalently to tissue macromolecules. Among the enzymes that metabolize xenobiotics, those associated with the cytochrome P-450-dependent monooxygenase system are probably the most important. The route by which a given substrate is metabolized in a tissue or cell is, generally, determined by the types and concentrations of cytochrome P-450 isozymes present. Understanding of enzyme distribution in the lung and appreciation of the species and cellular differences that exist is just beginning. (Auth. Abs. Mod.)

84–1772. Razma, A. G.; Lynch, J. P., III; Wilson, B. S.; Ward, P. A.; Kunkel, S. L. **Human Alveolar Macrophage Activation and DR Antigen Expression in Cigarette Smokers.** *Chest* 85(6, Supplement): 41S-43S, June 1984.

Alveolar macrophages (AMs) from smokers generated increased amounts of specific O_2 radical (superoxide anion), suggesting that smokers' AMs exist in a metabolically activated state and may have augmented potential to induce lung injury. AMs obtained by bronchoalveolar lavage from active smokers, former smokers, and nonsmoking controls were examined for surface expression of Ia-like (DR) antigen (Ag) and ability to generate the O_2 radical. The student's t test was used to compare control and experimental groups. The percentage of DR-bearing AMs is increased in smokers compared with nonsmokers. Although the role of AMs in the pathogenesis of emphysema is unclear, the finding of increased superoxide anion generation and increased expression of DR Ag on AM surface suggests that activated AMs may play significant roles in the evolution of this disease. (Auth. Abs. Mod.)

84–1773. Romert, L.; Bernson, V.; Pettersson, B. **Effects of Air Pollutants on the Oxidative Metabolism and Phagocytic Capacity of Pulmonary Alveolar Macrophages.** *Journal of Toxicology and Environmental Health* 12(2/3): 417-427, August-September 1983.

Data showed that the toxicity of extracts of airborne particles collected in rural and urban areas was infinitesimal compared with toxicity of cigarette smoke condensate (CSC). CSC, prepared from high-tar American-type cigarettes, was condensed and dissolved in dimethyl sulfoxide to a final concentration of 50 mg CSC/ml. Isolated rabbit pulmonary alveolar macrophages (PAMs) were used to investigate air pollutant toxicity. Valuable information on the interaction site and the mechanisms behind a toxic effect was obtained by studying phagocytosis and oxidative metabolism simultaneously. An extract of particles from a car tunnel was found to be the most toxic air sample, inhibiting phagocytosis and respiration of macrophages at a concentration representing 5 m^3 air/ml cell suspension. A corresponding sample collected on a roof of a five-story building in the central area of a city (population 600,000) was found to inhibit phagocytosis, but did not affect respiration. Further investigations revealed that one effect of the tunnel extract could be explained as an uncoupling of the mitochondrial respiratory control. The system used was sensitive enough to discriminate between various types of pollutants and reproducible enough to give a relevant dose—response correlation. (Auth. Abs. Mod.)

84–1774. Rylander, R.; Haglind, P. **Airborne Endotoxins and Humidifier Disease.** *Clinical Allergy* 14(1): 109-112, January 1984.

Symptoms of humidifier disease were more prevalent in nonsmokers than in smokers among 50 workers at a printing factory. Presence of the disease was investigated with particular emphasis on airborne endotoxins. Water in the humidifier was contaminated with *Pseudomonas*, and when the humidifier was operating, airborne endotoxin levels were 0.13 to 0.39 $\mu g/m^3$. Typical symptoms, reported by 20 workers, included fever, chills, and chest tightness when the humidifier was operating. The frequency of symptoms in nonsmokers may be related to the immune-depressive effect of tobacco smoke. The estimated inhaled dose of endotoxin was found to be sufficient to cause observed symptoms. The determination of airborne endotoxins in future episodes of humidifier disease is recommended. (Auth. Abs. Mod.)

84–1775. Slevin, N. J.; Aitken, J.; Thornley, P. E. **Clinical and Microbiological Features of *Branhamella catarrhalis* Bronchopulmonary Infections.** *Lancet* 1(8380): 782-783, April 7, 1984.

Of 101 patients with *Branhamella catarrhalis* bronchopulmonary infection, 94 were smokers, ex-smokers, or patients with chronic chest disease. These factors were more significantly associated with the infection than immunosuppression (59 patients) or high risk of oropharyngeal aspiration (17 patients). The pathogenicity of *B. catarrhalis* was evident from purulence of sputum, fever, blood leukocytosis, and patchy pulmonary shadowing on chest radiographs; the infection contributed to four of six deaths. *B. catarrhalis* was the only bacterial pathogen isolated from the sputum of 71 patients, and was isolated with other known pathogens from 30 patients. Suitable antibiotics included erythromycin, tetracycline, and co-trimoxazole; 38 of 99 isolates produced beta-lactamase.

84–1776. Stuart, B. O. **Deposition and Clearance of Inhaled Particles.** *Environmental Health Perspectives* 55: 369-390, April 1984.

Theoretical models of respiratory tract deposition of inhaled particles are compared with experimental studies of deposition patterns in humans and animals, as governed principally by particle size, density, respiratory rate, and flow parameters. Various models of inhaled particle deposition use approximations of the respiratory tract to predict fractional deposition caused by fundamental physical processes of particle impaction, sedimentation, and diffusion. These models for both total deposition and regional (nasopharyngeal, tracheobronchial, and pulmonary) deposition are compared with early and recent experimental studies. Reasonable correlation has been obtained between theoretical and experimental studies, but the behavior in the respiratory tract of very fine (<0.1 μm) particles requires further investigation. Properties of particle shape, charge, and hygroscopicity and the degree of respiratory tract pathology also influence deposition patterns; definitive experimental work is needed in these areas. The influence upon deposition patterns of dynamic alterations in inspiratory flow profiles caused by a variety of breathing patterns also requires further study, and the use of differing ventilation techniques with selected inhaled particle sizes holds promise in diagnosis of respiratory tract diseases. Mechanisms of conducting airway and alveolar clearance processes involving the pulmonary macrophage, mucociliary clearance, dissolution, transport to systemic circulation, and translocation via regional lymphatic vessels are discussed. (Auth. Abs.)

84–1777. Tabona, M.; Chan-Yeung, M.; Enarson, D.; MacLean, L.; Dorken, E.; Schulzer, M. **Host Factors Affecting Longitudinal Decline in Lung Spirometry Among Grain Elevator Workers.** *Chest* 85(6): 782-786, June 1984.

Cigarette smokers generally showed a greater decline in lung function than nonsmokers, but differences were not significant after adjusting for age. Host factors affecting the longitudinal decline in lung function were determined in 267 white male grain elevator workers who were still working in the industry and who did not change smoking habits over a 6-year period. At baseline, 25.8 percent were never smokers, 31.8 percent were ex-smokers, and 42.3 percent were current smokers. Spirometric measures declined more rapidly in older grain handlers compared with younger workers. Acute changes in lung function over the course of 1 workweek during the initial study were positively correlated with subsequent decline in lung function, as was bronchial hyperreactivity determined during followup. Positive immediate skin reactivity to common allergens, presence and absence of respiratory symptoms, and initial lung function did not appear to influence the subsequent decline in lung function. (Auth. Abs. Mod.)

84–1778. Tager, I. B.; Munoz, A.; Rosner, B.; Weiss, S. T.; Speizer, F. E. **Longitudinal Analysis of the Effects of Cigarette Smoking in Children.** *Chest* 85(6, Supplement): 8S, June 1984.

Lung function tests on 674 children aged 6 to 19 showed that relatively short periods of smoking could have [illegible]nd effects on the rate of increase in forced expiratory [illegible]ne in 1 sec (FEV_1). On any given year of the longitudinal study, 85 to 90 percent of the subjects were under age 14, yet frequency of smoking increased from 5.7 percent in year 4 to 9.9 percent in year 7. After adjustment for age, sex, height, and previous year's FEV_1, smoking significantly reduced the rate of increase of FEV_1. Lung function achieved by a smoking child after 2.5 and 5.0 years was predicted to be lower by 10 and 15 percent, respectively, compared with a nonsmoking child. At age 20, a subject who began smoking at age 15 would be expected to have an FEV_1 0.6 l lower than a comparable nonsmoker.

84–1779. Tashkin, D. P.; Clark, V. A.; Simmons, M.; Reems, C.; Coulson, A. H.; Bourque, L. B.; Sayre, J. W.; Detels, R.; Rokaw, S. **The UCLA Population Studies of Chronic Obstructive Respiratory Disease. VII. Relationship Between Parental Smoking and Children's Lung Function.** *American Review of Respiratory Disease* 129(6): 891-897, June 1984.

Analysis of questionnaire data and lung function tests on children revealed that passive exposure to maternal smoking affected airways of boys aged 7 to 11. A total of 971 white, non-Hispanic, nonsmoking, nonasthmatic children were divided into three categories related to parental smoking status: (1) at least mother smokes, (2) only father smokes, and (3) neither parent smokes. Prediction equations for several indexes of forced expired volume and flow were derived separately for boys and girls 7 to 17 years of age. Analysis of variance was performed separately on younger (aged 7 to 11) and older (12 to 17) children of each sex. Among younger male children, residual values were significantly lower in the maternal smoking category than in the other two household categories for maximal flow and maximal flow after exhalation of 25 percent of forced vital capacity (FVC) ($p \leq 0.05$); no differences were noted between the paternal smoking only and nonsmoking household categories. A trend toward similar results, which approached significance ($p<0.1$), was found in older male children. Among females, forced expiratory flow during the middle half of the FVC and maximal flow after exhalation of 75 percent of FVC were significantly lower in relation to maternal smoking in older children only ($p \leq 0.05$). ANOVA revealed no decrement in lung function in relation to passive smoking among children with asthma or bronchitis ($n=138$). Chi-square analysis showed no differences in the frequency of respiratory symptoms among children in the different passive exposure categories. The apparent effect on lung function of heavily exposed older girls is more likely to be confounded by selective underreporting of "active smoking." (Auth. Abs. Mod.)

84–1780. Taylor, R. G.; Gross, F.; Joyce, H.; Holland, F.; Pride, N. B. **Relation Between Bronchial Reactivity to Inhaled Histamine and Annual Decline in FEV$_1$ in Male Smokers and Exsmokers.** *Chest* 85(6, Supplement): 22S, June 1984.

To investigate the hypothesis that smokers with chronic airflow obstruction and asthmatics share a common allergic constitution and increased nonspecific bronchial reactivity, baseline forced expiratory volume (FEV$_1$) and annual decline of height-corrected FEV$_1$ (ΔFEV$_1$/ht^3) were studied in 117 smokers, 71 ex-smokers, and 39 nonsmokers. Of these, 29 percent of smokers, 24 percent of ex-smokers, and 5 percent of nonsmokers were bronchial reactors, requiring ≤ 16 mg/ml histamine to reduce FEV$_1$ by 20 percent. Reactors who smoked had a lower baseline FEV$_1$ and a faster ΔFEV$_1$/ht^3 than nonreactors, as did allergic smokers. Among ex-smokers, ΔFEV$_1$/ht^3 was similar in reactors and nonreactors, suggesting that enhanced bronchial reactivity acquired by smokers is not fully lost after quitting.

84–1781. The Tobacco Institute. **Cigarette Smoking and Chronic Obstructive Lung Diseases: The Major Gaps in Knowledge.** Washington, D.C., The Tobacco Institute, 1984, 14 pp.

It is argued that a causal relationship between smoking and either chronic bronchitis or emphysema has not been established scientifically, although the Surgeon General's annual reports have asserted that cigarette smoking is the principal cause of chronic obstructive lung disease (COLD). It is noted that (1) the causes of COLD are multiple and still poorly defined; (2) the trend of combining chronic bronchitis and emphysema makes it difficult to determine incidence and mortality and makes study data difficult to evaluate; and (3) there is scientific data that other factors may be related to onset of COLD. It is concluded that acceptance of smoking as the major risk factor for COLD may only discourage research needed to find the actual causes. The inconclusiveness of evidence against environmental tobacco smoke is also reviewed.

84–1782. Todisco, T.; Dottorini, M. **Mucus Hypersecretion, Airways Obstruction. (Letter).** *Lancet* 1(8380): 801, April 7, 1984.

Smoking had little influence on the prevalence of bronchial hyperreactivity (10.4 percent in smokers, 9.9 percent in nonsmokers) in an epidemiological study in Umbria, Italy. Nonspecific bronchial hyperreactivity to ultrasonic nebulized distilled water was present in 11.1 percent of 382 subjects selected from the normal working population. It is concluded that slow clearers (not bronchial reactors) will not acquire bronchorrhea or early bronchospasm, but are susceptible to small airways disease because of more prolonged action of noxious agents on poorly defended lungs. In contrast, fast clearers (bronchial reactors) have mucus hypersecretion as the main symptom. Mucociliary function and bronchial reactivity are useful indices of early, preventable risk.

84–1783. Weiss, S. T.; Speizer, F. E. **Increased Levels of Airways Responsiveness as a Risk Factor for Development of Chronic Obstructive Lung Disease. What Are the Issues?** *Chest* 86(1): 3-4, July 1984.

Competing risks may be why only 20 percent of cigarette smokers develop chronic obstructive lung disease (COLD). Smokers may die of coronary disease or lung cancer prior to developing COLD; however, susceptibility also may be an important factor. Two theories about the origin of COLD and study data testing the theories are reviewed. The British hypothesis holds that cigarette smoking leads to inflammation and phlegm production and abnormalities in airway clearance in persons susceptible to repeated infections that destroy lung tissue, producing a more rapid decline in lung function. The Dutch hypothesis holds that, in susceptible individuals, cigarette smoking leads to increased levels of bronchial responsiveness and eventually to a more rapid decline in pulmonary function. The major problem with existing longitudinal data is that only subjects with COLD have been examined. The important question is whether the causes of increased levels of responsiveness can be identified and whether such responsiveness is an independent risk factor for COLD. Because the issue is important, a variety of methodologic approaches should be considered.

84–1784. Weiss, T.; Dorow, P.; Felix, R. **Continuous Aerosol Inhalation Scintigraphy in the Evaluation of Early and Advanced Airways Obstruction.** *European Journal of Nuclear Medicine* 9(2): 62-67, February 1984.

Continuous aerosol scintigraphy showed reduction of bronchial mucus velocity to be one of the earliest signs of functional impairment in asymptomatic cigarette smokers. By continuous lung imaging, regional mucociliary removal rates of inhaled ^{99m}Tc-labeled human serum albumin minimicrospheres were determined over upper, middle, lower, central, and peripheral anterior lung areas. An index was employed for analysis of initial pulmonary radioaerosol distribution to quantify the site

of particle deposition in various degrees of airways obstruction. Participants included 15 healthy subjects, 20 asymptomatic smokers with early small airways obstruction, and 30 patients with advanced chronic obstruction of large airways. The healthy group showed evenly distributed lung activity and (normal) clearance rates consistent with earlier data. Although most uneven lung aerosol distribution was seen in patients with large airways obstruction, slowest mucociliary clearance velocity was encountered in the smokers with small airways dysfunction. (Auth. Abs. Mod.)

CARDIOVASCULAR DISEASES

84–1785. Beaumont, V.; Lemort, N.; Beaumont, J. L. **Evaluation of Risk Factors Associated With Vascular Thrombosis in Women on Oral Contraceptives. Possible Role of Anti-Sex Steroid Hormone Antibodies.** *Artery* 11(5): 331-344, 1983.

The presence of antiethinylestradiol (anti-EE) antibodies and smoking was significantly associated with idiopathic thrombosis in women using oral contraceptives (OCs), with the combined factors showing a synergistic influence. Cigarette use, age, anti-EE antibody levels, and serum lipid levels were analyzed in three groups of women: (1) 30 women who had never used OCs (controls); (2) 50 healthy OC users; and (3) 50 OC users with thromboembolic disease. There were more smokers in group 3 (50 percent) than in group 2 (34 percent) or group 1 (30 percent). Differences were significant when average consumption/day was considered; 9.5 vs. 4.6 and 3.3 cigarettes/day, respectively (34 percent in group 3 consumed >15 cigarettes/day compared with 16 percent in group 2). In group 2, 42 percent had no risk factors, smoking was the only risk factor in 28 percent, anti-EE the only risk factor in 24 percent, and only 2 percent had both anti-EE and smoking risk factors. In thrombosis patients, however, 6 percent had no risk factors, 4 percent had only smoking, 44 percent had anti-EE, and 46 percent had both smoking and anti-EE as risks.

84–1786. Burling, T. A.; Singleton, E. G.; Bigelow, G. E.; Baile, W. F.; Gottlieb, S. H. **Smoking Following Myocardial Infarction: A Critical Review of the Literature.** *Health Psychology* 3(1): 83-96, 1984.

This literature review strongly suggests that, although the actual percentages are debatable, a sizable proportion of individuals quit smoking following a myocardial infarction (MI) and that various types of advice to quit smoking may increase cessation rates. It also appears that post-MI smoking can seriously impact upon subsequent morbidity and mortality; for white males under the age of 70 who continue to smoke, rates of reinfarction and subsequent death are at least twice those of individuals who quit smoking. Other evidence suggests that mortality ratios of past studies may have underestimated the actual mortality due to smoking because of failure to control for baseline levels of MI severity. Studies conducted to date have methodological shortcomings, but it is argued that these studies as a whole have underestimated the impact of smoking resumption following MI, which may have occurred owing to (1) the inclusion of some smokers in the group of presumed quitters, and (2) the failure to control for a priori differences in medical severity/risk status between quitters and continuing smokers. If these problems were avoided, it seems likely that the health benefits of post-MI smoking cessation would appear even greater than generally reported. It is concluded that greater precision in study design and assessment procedures will reinforce and augment the present findings regarding the negative impact of continued smoking following MI. Further, improvement in design will aid in the determination of the factors (demographic, personality, social, and biological) that influence the decision to resume smoking or remain abstinent, which could lead to effective treatment interventions. (Auth. Abs.)

84–1787. Cairns, V.; Keil, U.; Kleinbaum, D.; Doering, A.; Stieber, J. **Alcohol Consumption as a Risk Factor for High Blood Pressure. Munich Blood Pressure Study.** *Hypertension* 6(1): 124-131, January-February 1984.

In an effort to separate the effects of smoking, alcohol, and other risk factors on elevated blood pressure, multivariate analysis of data from 3,198 participants (aged 30 to 69) in the Munich Blood Pressure Study was performed. Smoking was significantly interactive with body mass index, which could have been caused by overweight smokers with high blood pressure dying before they could be included in the study. Alcohol was a significant effect in most of the linear regression analyses for both men and women and for both systolic and diastolic blood pressure. The relationship between alcohol consumption and blood pressure was independent of cigarette smoking, age, sex, body mass index, oral contraceptive use, and years of education.

84–1788. Carlsson, I.; Wennmalm, A. **Platelet Aggregability in Smoking and Non-Smoking Subjects.** *Clinical Physiology* 3(6): 565-571, December 1983.

Data indicate that smokers differ from nonsmokers with regard to platelet function; platelet aggregability in response to arachidonic acid (AA) is unaffected, while the endogenous antiaggregatory power in the plasma is decreased. To investigate the hypothesis that the AA system has a different impact on platelet function in smoking compared with nonsmoking subjects, arterial blood was sampled from smokers and nonsmokers and platelet-rich plasma (PRP) was prepared. There were no differences in sex and age distribution between the groups. One portion of the PRP was used to determine the lowest amount of AA required to induce platelet aggregation. In other portions the endogenous antiaggregatory activity in the blood was determined (after reinforcing it with theophylline). There was no difference between smokers and nonsmokers in the amount of AA required to induce platelet aggregation. In fresh PRP prepared from blood from nonsmoking subjects, theophylline (10^{-4} M) induced a 12 to 17 percent inhibition of the ADP-induced aggregation of platelets, indicating the presence of endogenous subthreshold concentrations of PGI_2-like activity in the blood. The corresponding inhibition in fresh PRP prepared from blood from smokers was significantly lower (4 to 7 percent), suggesting lower endogenous concentrations of PGI_2-like activity, or decreased platelet sensitivity to the effects of such activity. The observations may be of significance for the cardiovascular hazards connected with smoking. (Auth. Abs. Mod.)

84–1789. Dacey, K. A. **Lidocaine Hydrochloride and the Pressor Response to Endotracheal Intubation: Comparison Study of Smokers and Non-Smokers.** Masters Thesis, California State University, Department of Nursing, Long Beach, University Microfilms International 83-21041, 1983, 59 pp.

The pressor response to endotracheal intubation was studied in 10 smokers and 10 nonsmokers who underwent surgery and general anesthesia. Two percent lidocaine hydrochloride, in a dosage of 1.5 mg/kg, was given i.v. 1 to 2 minutes prior to intubation in an attempt to blunt this response. Mean arterial pressure and heart rate were measured. The pressor response was seen in both groups. Nonsmokers manifested significant increases in mean arterial pressure at 0.5, 1.0, 2.0, and 3.0 minutes after intubation. No significant changes were seen in heart rate in this group. Smokers demonstrated significant elevations in both mean arterial pressure and heart rate at 0.5, 1.0, and 2.0 minutes after intubation. Mean arterial pressure increases were 5 to 14 percent higher among smokers. (Auth. Abs. Mod.)

84–1790. De Lorgeril, M.; Reinharz, A. **Possible Role de la Vasopressine Dans les Accidents Ischemiques Lies a la Consommation de Tabac. [Possible Role of Vasopressin in the Ischemic Complications of Smoking.]** *Archives des Maladies du Coeur et des Vaisseaux* 77(1): 71-75, January 1984, French.

In 12 of 25 patients, inhalation of tobacco smoke caused an increase in plasma vasopressin and nicotine-stimulated neurophysin (NSN, used as a marker of vasopressin secretion). Mean levels of NSN were 280±54 pg/ml before smoking and 714±459 pg/ml after smoking. Simultaneous measures of vasopressin and NSN every 5 min in four subjects confirmed the parallel changes of the two substances during smoking. Since smoking increases the risk of myocardial infarction and sudden death, and low doses of vasopressin (used for diagnosis and therapy) may induce ischemic complications in patients with coronary artery disease, it is suggested that vasopressin may play a primary role in the acute ischemic complications of tobacco smoking, and the "vasopressin response" could be used as a biological parameter to identify subjects at high cardiovascular risk from smoking. (Auth. Abs. Mod.)

84–1791. Epstein, F. H. **Lessons From Falling Coronary Heart Disease Mortality in the United States.** *Postgraduate Medical Journal* 60: 15-19, January 1984.

The marked decline in coronary heart disease (CHD) mortality in the United States since the late 1960s is probably due to successful efforts at primary prevention, attributable in turn to improved eating habits, better control of blood pressure, and a reduction in smoking. The number of current smokers over 20 years of age declined by about 25 percent among men and 14 percent among women between 1965 and 1980, serum cholesterol average levels have decreased, and hypertension has been detected and treated more efficiently. Another part of the decline can probably be explained by an improvement in prognosis. It remains to be established whether there has been a reduction in morbidity as well. Other countries have experienced declines (notably Australia, New Zealand, and Israel) to a lesser degree, but several countries have recorded increases in CHD mortality. These downward and upward turns, in general, are compatible with the theory that they are caused by corresponding favorable or unfavorable changes in lifestyles. (Auth. Abs. Mod.)

84–1792. Feinleib, M. **Changes in Cardiovascular Epidemiology Since 1950.** *Bulletin of the New York Academy of Medicine* 60(5): 449-464, June 1984.

The decline in smoking since the first Surgeon General's report in 1964 may be one of the factors responsible for the 38 percent decline in cardiovascular disease mortality since 1950; the reduction for all other causes was about 21 percent. The most remarkable trend is in age-adjusted mortality from coronary heart disease (CHD) that abruptly reversed (during the mid-1960s) the previous upward trend. Since 1968, CHD mortality has fallen 27 percent, reflected in 280,000 fewer premature deaths in the following decade than would have been expected on the basis of 1968 rates. However, it is still impossible to say with certainty why such a decline is occurring. Possible factors include problems related to data analysis and interpretation. Two phenomena that may also be at work (either a change in the incidence of CHD or a change in the case fatality rate) are discussed. Additional factors may be earlier and improved diagnosis, improved prehospital and hospital treatment, and availability of a wide variety of surgical and medical services. Available data suggest that improvement through prevention of controllable risk factors (smoking, high cholesterol, high blood pressure, diabetes mellitus, and obesity) may play a role. When trends in smoking, cholesterol, and blood pressure are translated into mathematical equations and related to the risk of dying from heart disease, the following predictions can be made: (1) a 5 mg/dl drop in mean cholesterol level would be expected to produce a 4.3 percent drop in the mortality rate; (2) a 2 mm Hg drop in diastolic blood pressure would result in an 8.7 percent decline; and (3) a 20 percent decline in cigarette smoking would have resulted in a 10 percent decline in mortality. If all three occurred, and there is some indication that they did, a 22 percent decline might be expected, almost exactly what was observed during the period to which the data apply. Among the goals for the year 1990 in the United States is that the proportion of adults who smoke will be reduced to below 25 percent.

84–1793. Fletcher, P. J. **Baroreceptor Heart Rate Reflex in Rabbits After Reversal of Renal Hypertension.** *American Journal of Physiology* 246(2, Part 2): H261-H266, February 1984.

Nasal smoke inhalation, elicited by blowing cigarette smoke at the external nares for 5 sec, produced similar degrees of reflex bradycardia in renal-wrapped (62±4 beats/min) and in sham-operated (59±6 beats/min) rabbits in a study of 26 hypertensive and 17 normotensive animals. In 13 rabbits undergoing reversal of hypertension, heart rate was similar before (59±6 beats/min) and after (50±4) unwrapping. Blood pressure and heart rate were varied by bolus injections of methoxamine or nitroglycerine in conscious rabbits, and a logistic sigmoid function was fitted to the pressure-heart rate data to estimate upper and lower heart rate plateaus, median blood pressure, and reflex sensitivity. In hypertensive rabbits, resetting of the blood pressure-heart rate curves to higher pressures was associated with reduced reflex sensitivity, increased lower heart rate plateau, and operating pressures significantly greater than the region of maximum reflex sensitivity, resulting in substantial attenuation of reflex bradycardia in response to hypertension. Reversing the hypertension completely reversed the abnormalities in baroreflex control of heart rate. Renal hypertension in the rabbit produces functional changes that are completely reversible in the baroreceptor-heart rate reflex, probably in the afferent limb. (Auth. Abs. Mod.)

84–1794. Gallavan, R. H., Jr.; Tsuchiya, Y.; Jacobson, E. D. **Effects of Nicotine on Canine Intestinal Blood Flow and Oxygen Consumption.** *American Journal of Physiology* 246(2, Part 1): G195-G203, February 1984.

In six series of experiments with adult mongrel dogs, intravenous infusion of nicotine at doses corresponding to those experienced by smokers produced a transient increase in systemic arterial blood pressure and mesenteric blood flow. Subsequently, a steady-state response developed that consisted of a reduction in mesenteric blood flow due to a decrease in blood pressure and an increase in intestinal vascular resistance. This increase in resistance was probably due to increased levels of circulating catecholamines. The intra-arterial infusion of nicotine into the intestinal circulation at doses experienced by the average smoker had no effect on either intestinal blood flow or oxygen consumption. Similarly, under in vitro conditions, nicotine had no direct effect on intestinal vascular smooth muscle tension. Thus, nicotine appears to reduce intestinal blood flow indirectly owing to systemic effects. (Auth. Abs. Mod.)

84–1795. Gulati, S. M.; Saha, K.; Kant, L.; Thusoo, T. K.; Prakash, A. **Significance of Circulatory Immune Complexes in Thromboangiitis Obliterans (Buerger's Disease).** *Angiology* 35(5): 276-281, May 1984.

Results of an immunological study suggest that significant quantities of biologically active circulatory immune complexes in sera from thromboangiitis obliterans (TO) patients are associated with smoking. Circulatory immune complexes were estimated in the sera of 25 TO patients and 20 age- and sex-matched controls (10 smokers, 10 nonsmokers) by precipitation with 8 percent polyethylene glycol (PEG). The immune complexes were characterized using the double immunodiffusion method. Mean protein content of the PEG precipitates was significantly higher in patients than in controls. Protein concentrations ranged from 380 to 2,940 μg/ml in control nonsmokers and from 1,280 to 5,400 μg/ml in control smokers ($p<0.05$). In TO cases, values were from 1,050 to 21,350 μg/ml, and 40 percent were over 4,000 μg/ml. Immunoglobulins in the immune complexes were predominantly of mixed type, in contrast with mainly single class immunoglobulins in controls. C3 complement distribution in immune complexes of patient and control sera was 64 and 45 percent, respectively. The immune complexes in patients were found to be biologically active. The mechanism of formation of circulatory immune complexes and their role in the pathogenesis of arteritis in relation to smoking is postulated. (Auth. Abs. Mod.)

84–1796. Gvozdjakova, A.; Bada, V.; Sany, L.; Kucharska, J.; Kruty, F.; Bozek, P.; Trstansky, L.; Gvozdjak, J. **Smoke Cardiomyopathy: Disturbance of Oxidative Processes in Myocardial Mitochondria.** *Cardiovascular Research* 18: 229-232, 1984.

Long-term cigarette smoking causes significant metabolic and morphological alterations to the heart muscle that can be characterized as smoke cardiomyopathy. The effect of cigarette smoke inhalation on oxidative and phosphorylative processes of rabbit myocardial mitochondria was studied in three experimental models after (1) a single 30-min inhalation, (2) 2 weeks smoking twice/day, and (3) 8 weeks smoking twice/day. Significant decreases in respiration and phosphorylation rates of mitochondria were found, but the respiratory control index and coefficient of phosphorylation remained unchanged. Both carbon monoxide and nicotine participate in the metabolic injury. (Auth. Abs. Mod.)

84–1797. Hart, J. T. **Hypertension and the Prevention of Coronary Heart Death in General Practice.** *Postgraduate Medical Journal* 60: 34-38, January 1983.

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Although it is accepted that coronary heart disease (CHD) is not caused by one identifiable factor, it must also be understood that prevention is multifactorial; control of smoking, dietary fat and obesity, and dietary sodium and potassium is important in addition to hypertension control. Results of randomized trials indicate community programs for blood pressure control have dramatic effects on reduction in stroke incidence. In Glyncorrwg (Wales), all adults have been screened for smoking, blood pressure, height, and weight since 1968. Risks are assessed individually and advice personalized for each patient. Smoking in both hypertensives and others has been reduced by about 50 percent: from 55 to 25 percent in hypertensive men and from 33 to 15 percent in hypertensive women. It is concluded that control of hypertension in British general practice is better than for any other CHD risk factor, with planned attacks on smoking and other factors having hardly begun. The need for Government support is emphasized.

84–1798. Heller, R. F.; Chinn, S.; Tunstall Pedoe, H. D.; Rose, G. **How Well Can We Predict Coronary Heart Disease? Findings in the United Kingdom Heart Disease Prevention Project.** *British Medical Journal* 288(6428): 1409-1411, May 12, 1984.

The probability of myocardial infarction developing over 5 years in 8,147 middle-aged men was predicted on the basis of age, blood pressure, cholesterol concentration, and smoking habit, recorded in an initial screening examination. Although the top 15 percent of the risk distribution predicted 115 (32 percent) of the subsequent cases of myocardial infarction, there was considerable overlap in predicted risk between subjects who did and those who did not go on to develop a myocardial infarction. Among subjects in the top 15 percent of risk, only 72 (7 percent) of those initially free of coronary heart disease and 43 (22 percent) of those initially with coronary heart disease actually developed a myocardial infarction over the subsequent 5 years. Thus, although a group of subjects at high risk can be identified, among whom will be a high proportion of potential victims of heart attack, many subjects will be wrongly classified. Findings may explain part of the difficulty in persuading patients of the potential benefits of coronary risk reduction. (Auth. Abs. Mod.)

84–1799. Hillbom, M.; Kaste, M. **Ethanol Intoxication: A Risk Factor for Ischemic Brain Infarction.** *Stroke* 14(5): 694-699, September-October 1983.

Although not generally regarded as a risk factor for brain infarction, smoking was more common among patients having ethanol-related brain infarction than in those whose infarction was not preceded by ethanol intoxication. Of 100 patients (67 men, 33 women) with acute ischemic brain infarction, 64 were smokers; however, there were no significant differences between the smoking habits of heavy drinkers and other patients. It is noted that both cigarette smoking and ethanol have been reported to increase blood viscosity. In 40 cases, the onset of symptoms was preceded within 24 hours by ethanol intoxication. Ethanol intoxication preceding brain infarction was 4 to 7 times as common in men and 6 to 15 times as common in women as ethanol intoxication in the general Finnish population of the same age and sex. Heavy drinking was twice as common in men and five times as common in women as heavy drinking in the general Finnish population of the same age and sex; 19 of the patients were heavy drinkers. Both occasional ethanol intoxication and regular heavy drinking seem to carry an increased risk of ischemic brain infarction. The ethanol-induced risk was highest in middle-aged women and young men. (Auth. Abs. Mod.)

84–1800. Jarvis, M. J.; Russell, M.A.H.; West, R.; Borland, C.; Chamberlain, A.; Higenbottam, T.; Shipley, M.; Rose, G. **Carbon Monoxide Yield of Cigarettes and Its Relation to Cardiorespiratory Disease. (Letters).** *British Medical Journal* 288(6416): 566-567, February 18, 1984.

The validity of the data base used in a study of the risk of fatal coronary disease (*British Medical Journal* 287(6405): 1583-1586, November 26, 1983) is questioned, noting that data on carbon monoxide yields from the Government Chemist's report could not be reconciled with yields shown in the earlier report, derived from the same source. Other fundamental discrepancies in the data, which was used to show that in men who inhaled, the risk of fatal coronary heart disease over a 10-year period was 51 percent lower in smokers of high carbon monoxide cigarettes are discussed. The original authors justify use of brands outside the years 1967-1970 by noting that within a given brand, yields changed little between 1965 and 1972. The close agreement between the tar values quoted by the Tobacco Research Council and the Government Chemist suggests support for the cigarettes being of similar composition and thus probably of similar carbon monoxide yield. It is claimed, therefore, that it is unlikely that laboratory errors in classifying an individual's exposure to carbon monoxide could have produced the positive results reported. See also 84-0492.

84–1801. Jenkins, B.; Kent, S.; Mayberry, J. F.; Colbourne, G. **Patients' Evaluation of a Post-Myocardial Infarction Teaching Programme Administered by Nurses.** *Postgraduate Medical Journal* 60(700): 108-110, February 1984.

Prevalence of smoking was reduced from 56 percent to only 7 percent 3 weeks after 228 myocardial infarction patients were given a detailed information session by nurses that included a program for exercise, and discussed attitudes toward smoking, diet, and sexual intercourse. Patients were asked to assess the value of the sessions using questionnaires administered during convalescence. Between 76 percent and 91 percent considered the presentation necessary, and there was a significant improvement in their understanding of infarction. More than 80 percent found a booklet entitled "Recovering From Your Heart Attack" particularly helpful. The great majority of patients believed that more health education should be given at school and that members of the general public should be taught the techniques of cardiopulmonary resuscitation. Among patients interviewed 2 to 8 months following infarction, 17 percent admitted smoking. (Auth. Abs. Mod.)

84–1802. Kannel, W. B.; Doyle, J. T.; Ostfeld, A. M.; Jenkins, C. D.; Kuller, L.; Podell, R. N.; Stamler, J. **Optimal Resources for Primary Prevention of Atherosclerotic Diseases.** *Circulation* 70(1): 157A-205A, July 1984.

An overview of smoking's relationship to cardiovascular diseases is included in a revision of the 1970 report "Primary Prevention of the Atherosclerotic Diseases." Recent research is analyzed and updated recommendations are made at three levels of prevention: individual behavior, physician-oriented preventive medicine, and public health policy. Specific topics addressed include (1) trends in cardiovascular disease mortality; (2) recent developments in public policy; (3) current theories of atherogenesis; (4) pediatric origins of atherosclerosis; and (5) results of recent intervention trials. The current status of modifiable risk factors is examined in detail, including smoking, blood pressure, weight, physical inactivity, and others. The roles of lipoproteins, diabetes mellitus, host factors, and combinations of risk factors and the preclinical signs of compromised circulation are also discussed.

84–1803. Khaw, K.-T.; Barrett-Connor, E.; Suarez, L.; Criqui, M. H. **Predictors of Stroke-Associated Mortality in the Elderly.** *Stroke* 15(2): 244-248, March-April 1984.

Cigarette smoking was positively related and educational level of head of household negatively related to stroke mortality in both sexes, with the predominant effect in women, in a 9-year followup of 1,056 men and 1,051 women (aged 65 to 84) in a southern California community. Age-adjusted stroke mortality rates for nonsmokers were 2.5 for men and 2.6 for women; for ex-smokers, 4.0 and 5.0; and for current smokers, 4.3 and 4.9. Relative risks for current vs. nonsmokers were 2.0 and 3.9, respectively. Age was the most significant independent risk factor for stroke-associated mortality in both sexes. Systolic blood pressure was weakly but significantly associated with stroke mortality. No significant independent effect of cholesterol, fasting plasma glucose, or obesity was discernible. Both smoking and blood pressure are potentially remedial risk factors for stroke after age 65. (Auth. Abs. Mod.)

84–1804. Kincaid-Smith, P.; Owen, J.; Whitworth, J. A.; Walter, M. A.; Doyle, A.; Woods, R. A. **Glomerular and Vascular Changes in Malignant Hypertension.** *Clinical and Experimental Hypertension* A6(1/2): 471-491, 1984.

Smoke exposure for 4 hours, twice/day, did not influence the frequency of experimentally induced malignant hypertension (MH) lesions in rats. In separate experiments, the pathological effects of MH induction and saline loading after complete aortic ligation were analyzed in several rat strains. Also, the prevalence and nature of MH lesions in 2 kidney 1 clip model Sprague Dawley (SD2K1C) and New Zealand genetically hypertensive (GHR) rats, exposed or not exposed to cigarette smoke, were examined. In rats subjected to complete aortic ligation, there were significant positive relationships between mean arterial pressure, plasma renin activity, and grade of histological lesions. Saline loading, however, abolished all correlations except between renin activity and histological grading of lesions. Presence of secretory droplets in epithelial cells also correlated with MH. The SD2K1C rats showed a far greater frequency of MH than GHR rats. Exposure to cigarette smoke had no influence on the prevalence of MH in SD2K1C or GHR rats.

84–1805. Larsson, B.; Svardsudd, K.; Welin, L.; Wilhelmsen, L.; Bjorntorp, P.; Tibblin, G. **Abdominal Adipose Tissue Distribution, Obesity, and Risk of Cardiovascular Disease and Death: 13 Year Follow Up of Participants in the Study of Men Born in 1913.** *British Medical Journal* 288(6428): 1401-1404, May 12, 1984.

Cigarette use was a significant risk factor for all-causes mortality and for ischemic heart disease, but not stroke, in a prospective study of risk factors for ischemic heart disease in 792 54-year-old men selected by year of birth (1913) and residence in Gothenburg. A large number of anthropometric and other measurements were made in 1967. At 13-year followup, neither quintiles nor deciles of initial indices of obesity (body mass index, sum of three skinfold thickness measurements, waist or hip circumference) showed a significant correlation with any of the three end points. Statistically significant associations were found between the waist-to-hip circumference ratio and the occurrence of stroke ($p=0.002$), ischemic heart disease ($p=0.04$), but not all-causes mortality ($p=0.053$). This ratio, however, was not an independent long-term predictor of the end points when smoking, systolic blood pressure, and serum cholesterol concentration were taken into account. Although smokers were leaner than nonsmokers, smoking was unrelated to the waist-to-hip circumference ratio. Results indicate that in middle-aged men the distribution of fat deposits may be a better predictor of cardiovascular disease and death than the degree of adiposity. (Auth. Abs. Mod.)

84–1806. Nolan, P. N.; Luk, D. E.; Staszewska-Barczak, J. **Reflex Effects Evoked From the Parietal Pericardium in the Dog: Comparison With Responses From the Visceral Pericardium.** *Basic Research in Cardiology* 78(6): 654-664, November-December 1983.

Nicotine (from 20 to 100 μg) produced no change in blood pressure or heart rate in dogs when applied to parietal pericardium and evoked depressor responses when applied to the epicardium of the left ventricle; depressor effects were abolished by vagotomy. Bradykinin (0.01 to 6.0 μg) elicited reflex increases in blood pressure and heart rate upon application to both sites that were not affected by either vagotomy or phrenic nerve section, but were suppressed by bilateral sectioning of the upper thoracic white rami communicantes and stellectomy. It is concluded that sympathetic, but not vagal, afferent endings innervating the parietal pericardium are susceptible to chemical stimulation and suggested that the pericardial sympathetic pressor reflex is nociceptive in nature and can be activated when kinin formation occurs during pericardial inflammation. (Auth. Abs. Mod.)

84–1807. Puchmayer, V. **Smoking as a Risk Factor for the Development of Arterial Occlusive Disease.** Acta Universitatis Carolinae Medica, Monographia 105, Univerzita Karlova, Praha, Czechoslovakia, 1984, 136 pp.

A total of 982 males with arterial occlusive disease, 30 females with atherosclerosis obliterans, and 411 male and 50 female controls were surveyed for smoking habits and other vascular disease risk factors. Among occlusive disease patients there were more long-term smokers (31 to 40 years), with higher cigarette consumption/day and an earlier age of smoking onset. The control group had significantly more ex-smokers of >5 years abstinence. Significantly more ex-smokers had only occlusions, and fewer had both occlusions and stenoses. Smoking >16 cigarettes/day was the major risk factor, with a disease latency of 31 to 40 years. However, the latency period decreased with increasing daily consumption. Also significant were smoking onset before age 20, "rapid smoking" (consuming a cigarette within 6 minutes), and an increase in consumption (of about 10 cigarettes) 5 to 8 years prior to onset of claudication symptoms. Patients affected in upper limb arteries tended to consume significantly more tobacco than those affected in the lower and abdominal arteries. Females showed the same latency period as males, but consumed 6 to 10 fewer cigarettes/day. Occupational and other risk factors are also detailed, and the etiology and pathogenesis of occlusive arterial disease is discussed.

84–1808. Puska, P.; Nissinen, A.; Salonen, J. T.; Toumilehto, J. **Ten Years of the North Karelia Project: Results With Community-Based Prevention of Coronary Heart Disease.** *Scandinavian Journal of Social Medicine* 11(3): 65-68, 1983.

Results of the 1982 followup study in North Karelia show that smoking and serum cholesterol levels continued to decline during the second 5-year period. Since 1972, smoking has been reduced 28 percent among men and 14 percent among women. Findings support the initial hypothesis that a well-conceived community-based program can have a beneficial impact on lifestyles and cardiovascular risk factors of an entire population and that such a development is associated with reduced cardiovascular mortality rates. Age-standardized coronary heart disease mortality among middle-aged males decreased by 24 percent compared with 12 percent nationally ($p<0.05$). The potential of a community-based approach for changing risk factors is demonstrated, affording practical experience in organizing such activities, which can be important for national and international demonstration and training purposes, within health services and other community settings. It is also important that risk factors and disease trends be monitored to assess the program's long-term impact. The program has been enlarged to consider related major noncommunicable diseases and to emphasize the promotion of "positive" health. The North Karelia project continues to apply its experience on a nationwide basis through the national health service system, several mass-media channels, and preparation of health education materials. There has also been active collaboration with the World Health Organization to help apply experience and results elsewhere.

84–1809. Rogers, R. L.; Meyer, J. S.; Shaw, T. G.; Mortel, K. F.; Thornby, J. **The Effects of Chronic Cigarette Smoking on Cerebrovascular Responsiveness to 5 Per Cent CO_2 and 100 Per Cent O_2 Inhalation.** *Journal of the American Geriatrics Society* 32(6): 415-420, June 1984.

Vasodilator capacitance to 5 percent CO_2 inhalation was reduced among 13 smokers compared with 16 nonsmokers of the same age by 48 percent in nonrisk subjects and 56 percent in subjects at risk for stroke, while vasoconstrictor capacitance to 100 percent O_2 inhalation among 64 smokers was decreased by 24 percent in nonrisk subjects and 34 percent in subjects at risk. Effects of chronic cigarette smoking on cerebrovascular responsiveness of 180 volunteers at risk and not at risk for stroke were evaluated by serial measurements of cerebral blood flow using the ^{133}Xe inhalation method. Resting gray matter blood flow values (Fg) measured while breathing room air were compared with Fg values measured during inhalation of either 5 percent CO_2 in air or 100 percent O_2. Changes in Fg values during inhalation of 5 percent CO_2 were used to estimate cerebral vasodilator capacitance, and those during inhalation of 100 percent O_2 were used to estimate cerebral vasoconstrictor capacitance. Results indicated that chronic cigarette smokers have both reduced vasodilator ($p<0.01$) and reduced vasoconstrictor ($p<0.02$) capacitance when compared with nonsmokers of the same ages regardless of whether or not other risk factors for stroke were present. In subjects at risk, combined effects of smoking and other risks appeared to be additive. (Auth. Abs. Mod.)

84–1810. The Tobacco Institute. **Cigarette Smoking and Heart Disease.** Washington, D.C., The Tobacco Institute, 1983, 52 pp.

Data from the Framingham and Oslo intervention trials and other studies on smoking as a risk factor for heart disease are cited as evidence that a relationship has not yet been established. It is suggested that the reduced risk in smokers who quit may be related to differences in a number of physical symptoms, personal characteristics, and miscellaneous traits between quitters and continuing smokers. Time trends in mortality rates are also not consistent with patterns of smoking and are difficult to explain, partly due to lack of adequate data. Geographical differences in disease incidence and mortality are also not consistent with the proposed smoking association. Some evidence exists that heredity plays a role in heart disease risk, and dietary factors have also been related to disease development. Behavioral and psychosocial factors such as personality and stress are also noted. It is concluded that no mechanism by which cigarette smoke might produce or contribute to heart disease has been demonstrated.

84–1811. Valora, N.; Bruno, C.; Mei, V. **Effetti Della Nicotina Sull'Elettrocardiogramma del Ratto. [Effects of Nicotine on the Electrocardiogram of the Rat.]** *Bollettino Societa Italiana Biologia Sperimentale* 59(6): 839-843, June 30, 1983, Italian.

Rats injected with nicotine (20 to 900 µg/kg, i.v.) exhibited electrocardiographic changes. At low doses, a conspicuous decrease in heart rate and an immediate, short-lived increase in a-v conduction time was seen. After 25 to 30 minutes, heart rate increased. At higher doses, heart rate was always increased. No extrasystoles were found, but occasional subendocardial damage or fleeting right bundle branch block was observed. At maximum dose (900 µg/kg), a marked but brief sinus node inhibition and a-v conduction block occurred. (Auth. Abs. Mod.)

84–1812. Watkins, L. O. **Why Are Death Rates From Coronary Heart Disease Decreasing? Which Preventive Measures Are Effective?** *Postgraduate Medicine* 75(8): 201-214, June 1984.

Declines in annual per capita cigarette consumption since 1963, diet modification, blood pressure control, and aerobic exercise have helped reduce the incidence of coronary disease. Increased prevalence of smoking in the 1950s and the decrease in the 1970s could have contributed to the increased coronary heart disease mortality in the 1950s and to recent decreases. However, although there was a proportionally greater decline between 1964 and 1975 in the percentage of male smokers compared with female smokers, the decline in mortality among women slightly exceeded that among men. The efficacy of primary prevention of coronary artery disease by multiple risk factor modification has been tested in large-scale clinical trials. Among middle-aged, normotensive, hypercholesterolemic smokers, smoking cessation and reduction of cholesterol levels by dietary change are followed by a decreased incidence of coronary events. Groups at high risk because of the combination of hypertension, cigarette smoking, and hypercholesterolemia show reduced coronary disease mortality after drug treatment of hypertension combined with cessation or reduction of cigarette smoking and dietary reduction of cholesterol levels. Primary prevention may be more effective if efforts begin early in life before behaviors and physiologic characteristics that may increase risk become established. (Auth. Abs. Mod.)

84–1813. Youkey, J. R.; Clagett, G. P.; Rich, N. M.; Brigham, R. A.; Orecchia, P. M.; Salander, J. M. **Femoral Anastomotic False Aneurysms. An 11-Year Experience Analyzed With a Case Control Study.** *Annals of Surgery* 199(6): 703-709, June 1984.

Continued tobacco use following aortofemoral bypass (AFB) was found to be a major risk factor for development of anastomotic false aneurysms due to host vessel degeneration. In 51 patients (all male smokers), 83 false aneurysms occurred from 1972 to 1982. All were heavy smokers (mean pack years 63±28), and 42 (82 percent) continued smoking after AFB. Of the 83 false aneurysms, 22 presented with acute events. Seventy (84 percent) were confirmed by sonography and/or arteriography. Bilaterality (29 percent), recurrence (18 percent), and associated false aneurysms (8 percent) were significant problems. Host vessel degeneration with an intact suture line remaining attached to a disrupted Dacron limb was the most common cause (61 percent) of false aneurysm. A case—control study of 23 cases with host vessel degeneration and 23 controls, matched for age, sex, and date of AFB compared cigarette smoking, hypertension, cerebrovascular disease, coronary artery disease, diabetes mellitus, previous femoral endarterectomy, outflow disease, other aneurysmal disease, multiple groin operations, wound complications, and training levels of surgeons performing initial AFBs. All were similar in cases and controls. Significant risk factors for false aneurysms were continued smoking after AFB ($p<0.005$), serum lipid abnormalities ($p<0.05$), and braided synthetic suture material as opposed to monofilament polypropylene ($p<0.05$). (Auth. Abs. Mod.)

84–1814. Zahavi, I.; Chagnac, A.; Hering, R.; Davidovich, S.; Kuritzky, A. **Prevalence of Raynaud's Phenomenon in Patients With Migraine.** *Archives of Internal Medicine* 144(4): 742-744, April 1984.

Smokers were slightly less likely to have Raynaud's phenomenon (RP) than nonsmokers in a prevalence study of 111 migraine patients (25 smokers) and 111 age- and sex-matched controls (26 smokers). Among smokers, 6 of 51 (12 percent) and only 1 of 11 heavy smokers (>20 cigarettes/day) had RP compared with 28 of 161 nonsmokers (17 percent). RP was diagnosed in 26 percent of migraine patients (33 percent of 39 patients with classic and 22 percent of 72 patients with common migraine) and in 7 percent of controls. Drug treatment did not account for the difference in prevalence. It is concluded that RP occurs more frequently in migraine patients than in the general population.

See also, **84–1702, 84–1850, 84–1870**

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PREGNANCY AND INFANT HEALTH

84–1815. Abel, E. L. **Marihuana, Tobacco, Alcohol and Reproduction.** Boca Raton, Florida, CRC Press, 1983, 254 pp.

Research data about the effects of tobacco, alcohol, and marihuana on sex and reproduction in humans and animals are summarized. Information on smoking-related effects includes (1) the pharmacology of cigarette smoke components; (2) prevalence of smoking among adults of childbearing age; (3) tobacco use and sex, including the effect of smoking on male sexuality, sperm, sex steroids, and female reproductive physiology; (4) tobacco's effects on growth and development (spontaneous abortion, birth weight, perinatal mortality, malformations, Apgar scores, long-term growth and behavior, respiratory disorders, sudden infant death, and teratogenicity); and (5) mechanisms by which smoking affects fetal development (maternal weight and constitution, chromosomal abnormalities, placental changes, fetal breathing activity, cyanide toxicity, carboxyhemoglobin, and lactation). The use of marihuana and alcohol is also reviewed in detail and areas of similarity and differences between effects of the three substances are indicated. It is noted that some studies show smoking and alcohol consumption produce a greater impact than either substance used separately.

84–1816. Black, P. **Who Stops Smoking in Pregnancy?** *Nursing Times* 80(19): 59-61, May 9, 1984.

Study data on 367 pregnant women, and a review of the literature, show that individual support is essential to cessation of smoking during pregnancy. Knowledge alone, poster campaigns, or general media efforts do not influence smoking during pregnancy, while anecdotal evidence suggests that a lower weight baby means an easier delivery to women who smoke. Also, personal experience of a healthy baby born to a smoker will cause a mother to discount other evidence. Interviews with the 367 mothers (143 smokers) showed that 21 percent claimed they had never heard of the harmful effects of cigarettes during pregnancy, while 20 percent knew of smoking's effects, but chose to continue smoking. Only 9 percent quit smoking while pregnant, although 48 percent attempted and 40 percent succeeded in reducing consumption. Nearly half of those who quit did so because smoke became distasteful during pregnancy. The most important factor in reducing overall consumption, however, was a personal warning given by a medical advisor.

84–1817. Cummings, A. J.; Kaelin, A. C. **A Study of the Effect of Cimetidine on Human Placental Mono-Oxygenase Activity *In Vitro*.** *Biochemical Society Transactions* 11(2): 178-179, April 1983.

Data indicated that cimetidine has a weak inhibitory effect on smoking-induced monooxygenase activity in human placenta. Placentas from women who smoked during pregnancy had greatly increased activity of cytochrome P448 monooxygenase enzyme systems. The concentrations of cimetidine required for 50 percent inhibition of activity were 100-fold or more than the plasma concentrations obtained from usual therapeutic doses. The effects of cimetidine resemble those of metyrapone, which is thought to inhibit cytochrome P-450-associated monooxygenases, rather than α-naphthoflavone that specifically inhibits cytochrome P-448-associated enzymes.

84–1818. Dalaker, K.; Grunfeld, B.; Jansen, A. **Smoking in Pregnancy.** *Journal of the Oslo City Hospitals* 34(3/4): 21-27, March-April 1984.

Data for [illegible]427 births confirm reports that the mean birth weight of infants of mothers who smoke is lower than for infants of nonsmokers. The frequency of low birth weight infants (<2500 g) was 75 percent higher among smokers (n=905). The importance of smoking was independent of other social factors, although there was also a positive correlation between low birth weight and maternal age below 20 years, unmarried status, poor housing conditions, and low educational level. Mean gestational age was somewhat shorter among smokers, calculated from date of last menstrual period, but no difference was found at clinical estimation of gestational age. Perinatal mortality was significantly more common among smokers, particularly stillbirths caused by abruptio placentae. The total proportion of complications of pregnancy and delivery was the same for smokers and nonsmokers.

84–1819. Gal, P.; Sharpless, M. K. **Fetal Drug Exposure—Behavioral Teratogenesis.** *Drug Intelligence and Clinical Pharmacy* 18(3): 186-201, March 1984.

In a review of the neurobehavioral and developmental effects of fetal drug exposure, several studies are cited that indicate maternal smoking as a cause of a number of adverse effects on the fetus and newborn, including increased perinatal mortality, growth retardation affecting height and weight (but not head circumference), congenital anomalies, low Apgar scores, and neurologic and developmental problems. Investigations of long-term effects have produced conflicting results, but there is some evidence of a significant relationship between increased consumption of cigarettes during pregnancy and increased hyperactivity, low achievement, and soft neurologic signs in 7-year-old children. Other drug exposures discussed include thalidomide, alcohol, marihuana, anticonvulsants, psychotropic drugs, narcotics, antibiotics, anticoagulants, drugs used in cancer therapy, cardiovascular drugs, corticosteroids, sex steroids, vaginal spermicides, gastrointestinal drugs, thyroid and antithyroid therapy, and environmental toxins. The difficulty of providing conclusive data due to complicating factors makes large epidemiologic studies and animal research necessary. Clinicians need to weigh the validity of available data to determine which drugs will cause behavioral and developmental sequelae following antenatal exposure and, consequently, which children will need more careful developmental evaluation and followup. Behavioral teratogenesis should be considered when selecting drug therapy during pregnancy. (Auth. Abs. Mod.)

84–1820. Hauth, J. C.; Hauth, J.; Drawbaugh, R. B.; Gilstrap, L. C., III; Pierson, W. P. **Passive Smoking and Thiocyanate Concentrations in Pregnant Women and Newborns.** *Obstetrics and Gynecology* 63(4): 519-522, April 1984.

No evidence was found that passive cigarette smoke exposure resulted in higher maternal or umbilical cord thiocyanate concentrations than found in nonsmokers in an investigation of 29 pregnant women who smoked, 51 who were passively exposed (lived or worked with a smoker), and 83 nonsmokers. Mean thiocyanate concentration (95 μmol/l) was significantly higher ($p<0.001$) in smokers than in passive smokers (35.9 μmol/l) or nonsmokers (32.3 μmol/l). Maternal and umbilical mean cord thiocyanate concentrations were similar in the smoking group (95 vs. 72 μmol/l). Although umbilical cord levels in infants of passive smokers and nonsmokers were similar (26 vs. 23 μmol/l), both levels were significantly lower than in smokers. Most important, there was an inverse relationship between umbilical cord thiocyanate concentration and birth weight ($p<0.001$). Maternal weight gain during pregnancy and infant birth weights were similar for passive smokers and nonsmokers, but there was a significant relationship between umbilical cord thiocyanate concentration and birth weight in infants of smokers. (Auth. Abs. Mod.)

84–1821. Hogue, C.J.R. **The Effect of Common Exposures on Reproductive Outcomes.** *Teratogenesis Carcinogenesis and Mutagenesis* 4(1): 45-57, 1984.

Problems related to research on the multiple risks involved in pregnancy are discussed, noting that when data are confounded (e.g., lethality of acetaldehyde in combination with nicotine and caffeine in rats), analysis should include testing for both confounding and interaction, since a common exposure (nicotine) may be not only a confounding factor but also a synergistic agent. Such testing may reveal groups of persons at particularly high risk and provide clues as to mechanisms of action. Data have shown that in investigations of coffee drinking and low birth weight, cigarette smoking is a classic confounding factor. Measurement of exposures can create problems also, especially if it relies on maternal recall, which can be unreliable. In addition, sample size is a problem since the group sizes required to study modest level effect must be so large. Although common exposures produce complexity, they may also help unravel mysteries surrounding less common exposures.

84–1822. Madsen, H.; Ditzel, J. **Effect of Smoking on Red Cell Oxygen Transport and Release in Diabetic Pregnancy.** *Acta Obstetricia et Gynecologica Scandinavica* 63(1): 77-80, 1984.

Red cell oxygen transport and release data suggest that smoking in pregnant diabetic women impairs the adaptive increase in 2,3-diphosphoglycerate (2,3-DPG) associated with diabetes-induced hypoxia. Two groups of pregnant diabetics (23 smokers, 23 nonsmokers) were studied in the third trimester. Blood glucose regulation was comparable between groups, since median blood glucose and glycosylated hemoglobin concentrations did not differ. Red cell 2,3-DPG levels were significantly lower in smokers compared with nonsmokers (16.5 vs. 17.8 μmol/g Hb, $p<0.01$). Partial pressure indicating 50 percent oxyhemoglobin saturation (P_{50}) of the oxyhemoglobin dissociation curve at actual pH and at pH 7.40 was also significantly lower in smokers compared with nonsmokers (25.9 vs. 26.9 mm Hg, $p<0.01$, and 26.5 vs. 27.8 mm Hg, $p<0.01$, respectively). Red cell 2,3-DPG was significantly correlated with P_{50} at pH 7.40 ($r=0.73$, $p<0.001$). Arterial oxygen saturation was reduced to the same degree in smokers and nonsmokers compared with healthy nonsmoking pregnant women, and no adaptive increase in hemoglobin concentration occurred in the pregnant diabetic smokers. It is concluded that, if hypoxia is causative for increased risk of fetal intrauterine death in diabetes, pregnant diabetic women should be advised not to smoke. (Auth. Abs. Mod.)

84–1823. Martin, K. A. **An Assessment of Behavior in Infants of Women Who Smoke During Pregnancy.** Doctoral Dissertation, Boston College, Graduate School of Arts and Sciences, University Microfilms International 83-20351, 1983, 189 pp.

The neurobehavioral effects of smoking during pregnancy were assessed in infants of 10 heavy smokers (>15 cigarettes/day) using the Brazelton Neonatal Behavioral Assessment Scale and compared with infants of 10 nonsmokers. Infants of smokers had both significantly lower mean Apgar scores (7.5) and birth weights (3261.3 g) compared with nonsmokers' infants (8.6 and 3663.2 g, respectively). Nonsmokers' offspring were also slightly longer. Behaviorally, smokers' infants showed decreased orienting behavior, responded generally less well on cuddliness or to being held, and tended to show more marked color changes during examination as a physiological response to stress. Smokers' infants also differed on two items relating to motor processes of tone and motor capacity (use of hand-to-mouth facility and pull-to-sit maneuver). Four infants of smokers compared with only one from a nonsmoker showed suspicious neurological findings.

84–1824. McIntosh, I. D. **Smoking and Pregnancy: Attributable Risks and Public Health Implications.** *Canadian Journal of Public Health* 75(2): 141-148, March-April 1984.

Data from previous research in smoking and pregnancy outcome found that between 5 and 25 percent of unfavorable outcomes in all pregnancies may be attributable to smoking.

Data for a total of 260,000 women were reworked, and rates for pregnancy outcome variables in smoking and nonsmoking mothers were extracted. Odds ratios, relative risks, and 95 percent confidence limits were estimated for each variable, and the results tabulated. Of the 28 variables, 10 are related to the mother and 18 to the offspring. The proportion of women who smoked during pregnancy ranged from 14 percent in American Orientals to 61 percent in whites. Overall, approximately 42 percent smoked while pregnant. Smoking-related attributable risks varied from 1.2 (congenital abnormalities) to 56.7 (deaths, perinatal to 5 years), with half the values below 10.0. The proportion of unfavorable events occurring in smokers and attributable to smoking ranged from 3.1 percent to 43.4 percent. The validity of the attributable risk estimates presented are discussed in detail, as are the choice of study populations, comparisons with other studies, smoking as a causal factor, and the possibility for prevention.

84–1825. Meirik, O.; Nygren, K.-G. **Outcome of First Delivery After 2nd Trimester Two-Stage Induced Abortion. A Controlled Historical Cohort Study.** *Acta Obstetricia et Gynecologica Scandinavica* 63(1): 45-50, 1984.

A positive correlation ($p<0.005$) between maternal smoking and history of abortion was found in a historical cohort study of the outcome of the first birth after a legal second trimester two-stage abortion induced with saline or prostaglandin $F_{2\alpha}$. Birth outcome for 142 nulliparous and 65 parous aborters was compared with that for 180 parity-matched controls and for all Swedish women in 1975. No statistically significant differences were found in mean birth weight and length of gestation or in occurrence of low birth weight (LBW) infants and preterm deliveries. However, nulliparous women with a previously induced abortion tended to have more LBW infants than other nulliparae. More LBW infants were born to the 29 nulliparae with a previous prostaglandin-induced abortion than to the 113 nulliparae having abortions induced with saline ($p=0.11$). No other noteworthy differences were found in birth outcome for the 39 women with a previous abortion induced by prostaglandin compared with the 168 that had saline-induced abortions. It is concluded that the LBW of infants born to aborters may be related as much to maternal smoking as to the previously induced abortion. (Auth. Abs. Mod.)

84–1826. Ounsted, M.; Moar, V. A.; Cockburn, J.; Redman, C.W.G. **Factors Associated With the Intellectual Ability of Children Born to Women With High Risk Pregnancies.** *British Medical Journal* 288(6423): 1038-1041, April 7, 1984.

Results of a study comparing the intellectual abilities of 242 children born to women who had been hypertensive during pregnancy showed that, at age 7.5, memory was significantly ($p<0.05$) less developed and all other parameters in the British ability scale were slightly less among children of mothers who smoked. Associations between 15 maternal, fetal, perinatal, postnatal, and environmental factors and test scores were investigated. After adjustment for confounding variables, children in the upper social classes, born to nonsmokers, who were firstborn and breast fed and with birth weights above the 10th centile, had significantly higher scores in some aspects of ability than the rest. Children whose mothers had developed superimposed pre-eclampsia had higher scores then those whose mothers had not suffered pre-eclampsia, and children delivered by elective cesarean section had lower scores than those delivered spontaneously. In a small subgroup of women with particularly high risk pregnancies, perinatal mortality had been 10 times greater than in the rest of the sample. At 7.5 years the intellectual ability of the survivors did not differ from the rest. Findings do not support the notion that there is a quantitative continuum of "reproductive casualty" from mortality to morbidity. (Auth. Abs. Mod.)

84–1827. Philipp, K.; Pateisky, N.; Endler, M. **Effects of Smoking on Uteroplacental Blood Flow.** *Gynecologic and Obstetric Investigation* 17(4): 179-182, April 1984.

Effects of nicotine consumption on uteroplacental blood flow were studied in 20 pregnant women by means of placental blood flow measurements using indium-113m-transferrin. Smokers were found to have a higher rate of poor perfusion patterns than subjects of a control group. In addition, the number of cigarettes smoked daily was significantly correlated with the placental perfusion type. The mean birth weight of infants of smokers was 250 g lower than in controls. Results suggest that smoking during pregnancy may compromise uteroplacental blood flow and thus result in poor fetal development. (Auth. Abs.)

84–1828. Public Health. **Low Birth Weight, Drink, Smoking and Diet.** *Public Health* 97(6): 307-308, November 1983.

Although the association between maternal smoking and birth weight has received a good deal of attention, smoking and drinking are highly correlated and it is questionable whether the effects of tobacco and alcohol are independent. It is unlikely that either alcohol or tobacco use accounts for more than a minority of cases of low birth weight. Maternal nutrition at conception is significant, and nutritional deficiency is hard to correct during the first trimester, when nausea is common. Mineral status is also important; low birth weight has been associated with low total iron-binding capacity in early pregnancy, and leukocyte zinc level (but not serum zinc) has been strongly correlated. The interactions between bivalent metals and mineral metabolism during pregnancy awaits clarification.

84–1829. Rogozinski, H.; Ankers, C.; Lennon, D.; Wild, J.; Schorah, C.; Sheppard, S.; Smithells, R. W. **Folate Nutrition in Early Pregnancy.** *Human Nutrition: Applied Nutrition* 37A(5): 357-364, October 1983.

Folate intake was highest in nonsmokers ($n=118$) or those who smoked <10 cigarettes/day ($n=25$) among 195 women assessed during the first trimester of pregnancy. The difference in intake of free folate between nonsmokers and those smoking ≥ 10 cigarettes/day (n=52) was significant ($p < 0.05$); for total folate the difference between nonsmokers and those smoking 10 to 19 cigarettes/day was significant. Trends in folate intake were also associated with lower social class, maternal age under 20 years, and vomiting ≥ 3 days/week. Total folate intake ranged from 64.7 to 302.0 μg/day (mean intake 148.0 μg/day). Prior to the use of folate supplements in the second and third trimester, megaloblastic anemia of pregnancy was one of the commonest vitamin deficiency diseases in developed countries. Results indicate that young mothers of low social class (those with the lowest folate intake) may need supplements in the first trimester, when adequate folate provisions for the embryo may be important for neural tube closure.

84–1830. Staisey, N. L.; Fried, P. A. **Relationships Between Moderate Maternal Alcohol Consumption During Pregnancy and Infant Neurological Development.** *Journal of Studies on Alcohol* 44(2): 262-270, January 1983.

Alcohol consumption did not contribute to smoking-related differences in birth weight among the infants of the 59 women analyzed. Subjects, surveyed for smoking habits and alcohol consumption, had a mean age of 29.6±4.7 years, mean gravida of 2.3±1.4, and mean weight gain of 13.3±4.7 kg. Alcohol and nicotine consumption were related during pregnancy ($r=.30$, $p<0.01$), but not prior to pregnancy. Compared with prepregnancy levels, nicotine intake dropped to a third and alcohol intake to about half during pregnancy. When smoking was accounted for, the association between maternal alcohol consumption and infant birth weight disappeared. However, after controlling for maternal nicotine intake, alcohol consumption was negatively associated with infant muscle tone and length of labor, and positively associated with spontaneous startle reactions in the infant at 9 days of age. No relationship was found between maternal alcohol consumption and infant physical characteristics, motor activity, general reflex activity, or eye characteristics.

84–1831. Wright, J. T.; MacRae, K. D.; Barrison, I. G.; Waterson, E. J. **Effects of Moderate Alcohol Consumption and Smoking on Fetal Outcome.** In: *Mechanisms of Alcohol Damage In Utero.* Ciba Foundation Symposium 105, London, Pitman, 1984, pp. 240-253.

Results from previous studies on the effects of moderate alcohol consumption and cigarette smoking on pregnancy outcome are assessed. Consumption of more than 10 g alcohol/day in very early pregnancy or even before conception approximately doubled the risk of low birth weight. However, maternal smoking is also a risk factor for low birth weight, and many investigations of the effects of alcohol failed to adjust for smoking and other factors. It is noted that many women quit drinking during pregnancy but continue to smoke, and the effects of changes in such habits are difficult to establish. Even so, there appears to be no significant interaction between tobacco and alcohol, with total relative risk obtained by multiplying individual risks from smoking and drinking. Results of a prospective study of the association between alcohol consumption in pregnancy and birth weight, length of baby, head circumference, and premature delivery are described. (Auth. Abs. Mod.)

OTHER DISEASES AND CONDITIONS

84–1832. Barakat, M. H.; Menon, K. N.; Badawi, A. R. **Cigarette Smoking and Duodenal Ulcer Healing. An Endoscopic Study of 197 Patients.** *Digestion* 29(2): 85-90, May 1984.

Although healing rates in ulcer patients who smoked were slightly lower than those of nonsmokers, the differences were not significant (relative risk of nonhealing attributable to smoking was 1.12). The healing rate of duodenal ulcer was endoscopically studied in 82 heavy smokers and compared with a nonsmoking control group of 92 patients. The two groups received antiulcer treatment in a double-blind fashion with one of the following regimens: cimetidine 1 g, trimipramine 50 mg, prostaglandin E_1 analog 800 or 200 μg, low-dose antacid, or placebo. Smokers' overall healing rate was 45.1 percent compared with 51.1 percent in nonsmokers. The difference between the two groups was not statistically significant. This finding is not in agreement with previous reports showing an adverse effect of smoking on the healing process of duodenal ulcer. (Auth. Abs. Mod.)

84–1833. Carl, P.; Crawford, M.; Ravlo, O. **Tobaksforgiftning Hos Børn. [Tobacco Poisoning in Children.]** *Ugeskrift for Laeger* 146(15): 1160-1161, April 9, 1984, Danish.

During the period from 1974 to 1980, 24 children were hospitalized and 98 were treated on an outpatient basis after ingestion of tobacco. Symptoms of poisoning were few, and no cases of severe poisoning were observed. No cases of nicotine poisoning are mentioned in the literature. It is concluded that hospitalization in most cases is not necessary, but gastric aspiration is recommended if vomiting has not occurred. (Auth. Abs.)

84–1834. Demol, P.; Singer, M. V.; Hotz, J.; Dipp, M.; Goebell, H. **Interdigestive Gastrointestinal Motility and Secretion of Gastric Acid and Pancreatic Enzymes in Young Cigarette Smokers.** *Digestion* 29(2): 107-111, May 1984.

No significant overall changes were observed in determinations of the basal (interdigestive) gastrointestinal motor activity and the interdigestive gastric acid and pancreatic amylase secretion in 10 young smokers (mean age 23 years) and 7 nonsmokers (mean age 26 years). There was no significant difference in the length of the interdigestive motor complex between smokers and nonsmokers, but the speed of transmission of the activity front (phase III) in the upper intestine was significantly ($p < 0.05$) greater in smokers than in nonsmokers. Apparent contradiction with earlier findings of increased enzyme secretion in older smokers points to a possible time—dose-dependent effect of cigarette smoking. (Auth. Abs. Mod.)

84–1835. Domschke, S.; Domschke, W. **Gastroduodenal Damage Due to Drugs, Alcohol and Smoking.** *Clinics in Gastroenterology* 13(2): 405-436, May 1984.

Cigarette smoking is associated with an increased incidence of gastric and duodenal ulcers. It also impairs healing and favors relapse, although the factors involved have not been clearly identified. Cessation of smoking may be one of the best and cheapest adjuncts of treatment and prophylaxis, especially in duodenal ulcer patients. It has been shown that some drugs can cause acute gastroduodenal damage, but even for the most widely studied offender, acetylsalicylic acid, major bleeding or ulceration could be established only for frequent and heavy use. Glucocorticoids may enhance ulcer risk when administered at high dosage for prolonged periods to susceptible individuals. Chronic moderate alcohol consumption by itself does not seem to increase the liability to peptic ulceration. However, abstinence from smoking should be strongly recommended in view of other severe consequences of smoking, such as chronic respiratory disease, bronchial cancer, ischemic cardiovascular disease, and deleterious effects in pregnancy and to neonates. Although patients can seldom be motivated to cease smoking before serious sequelae occur, they may be more easily convinced if they suffer from repeated ulcer attacks. (Auth. Abs. Mod.)

84–1836. Eckel, R. H.; Sadur, C. N.; Yost, T. J. **Smoking, Weight Change, and Lipoprotein Lipase. (Letter).** *New England Journal of Medicine* 311(4): 259-260, July 26, 1984.

Additional data confirms earlier findings (*New England Journal of Medicine* 310(10): 614-616, March 8, 1984) of a relationship between lipoprotein lipase activity in adipose tissue and body weight changes in adult smokers. Lipase activity was similar in 8 smokers and 22 nonsmokers on hypocaloric diets (325-675 kcal/day, 8.0±1.0 vs. 13.7±2.0 nanoequivalents of free fatty acid/minute/10^6 cells, not significant). However, after 3 weeks, weight loss was related to baseline (fasting) lipoprotein lipase activity in adipose tissue among smokers but not nonsmokers. Smokers with high activity lost less weight. It is suggested that the ability of adipose-tissue enzyme activity to predict weight loss in dieters who smoke represents a smoking-related alteration in energy conservation. See also 84-1011.

84–1837. Epstein, L. H.; Dickson, B. E.; McKenzie, S.; Russell, P. O. **The Effect of Smoking on Perception of Muscle Tension.** *Psychopharmacology* 83(1): 107-113, April 1984.

Results showed that six male and six female smokers produced less muscle activity during high-arousal situations than in low-arousal situations and that smoking reduced sensitivity to muscle activity in females, but enhanced sensitivity in males. Subjects were exposed to four conditions to determine the effects of smoking on perception of muscle activity: high and low arousal crossed with smoking or deprivation. When compared with six male and six female nonsmokers, smokers in a deprived state generated more muscle activity during the perception task, but showed no differences in sensitivity. Results represent the initial demonstration that smoking can alter the perception of physiological processes. In a second study of 11 female smokers, designed to determine whether the effects of smoking were specific to muscle tension or indicative of a more generalized perceptual change, perception of muscle tension and decibel levels were compared in smoking or deprived smokers who were in the high-arousal situation. Results for muscle-tension perception in high-arousal situations were similar to those in the first study, but smoking did not alter perception of auditory stimuli. (Auth. Abs. Mod.)

84–1838. Goldzieher, J. W. **Hormonal Contraceptives. Past, Present, and Future.** *Postgraduate Medicine* 75(5): 75-77, 80, 83-84, 86, April 1984.

In a review of the possible risks and benefits of oral contraceptive (OC) use, it is noted that in studies that appeared to show a relationship between myocardial infarction (MI) or stroke and hormonal contraceptive use, cigarette smoking was a major confounding factor. Mortality data from a British study showed a total of 13 deaths in nonsmokers (9 current or former OC users) compared with 48 in smokers (42 current or former OC users). When results are adjusted for smoking, the risk of MI or stroke is almost exclusively in heavy smokers. It is concluded that OC use should be an option for all fertile women, since evidence to date points to protective rather than harmful effects. Research on improved formulations is reviewed, noting that an injectable agent for use once a month is in widespread use outside the United States and an implantable or injectable agent will probably be approved eventually for this country.

84–1839. Grunberg, N. E.; Bowen, D. J.; Morse, D. E. **Effects of Nicotine on Body Weight and Food Consumption in Rats.** *Psychopharmacology* 83(1): 93-98, April 1984.

Research designed to determine whether consumption of nonsweet food is affected by nicotine and whether continuous access to only nonsweet foods attenuates the body weight changes associated with nicotine administration and cessation of nicotine administration is described. Alzet miniosmotic pumps were implanted subcutaneously to administer saline or three different concentrations of nicotine to male Sprague-Dawley albino rats for 2 to 3 weeks. Two studies on a total of 80 rats found an inverse dose—response relationship between nicotine administration and body weight without changes in bland food or water consumption. After cessation of nicotine administration, there were no differences in food consumption or body weight changes between groups. The effects of nicotine on body weight, both during and after drug administration, were attenuated compared with results of studies that provided sweet-tasting foods. Findings have implications for control of the marked gains in body weight that plague many ex-smokers. (Auth. Abs. Mod.)

84–1840. Hedin, C. A.; Larsson, A. **The Ultrastructure of the Gingival Epithelium in Smokers' Melanosis.** *Journal of Periodontal Research* 19(2): 177-190, March 1984.

Results indicate that tobacco smoking may be a causative factor in melanin pigmentation of the oral mucosa and that smoking activates the epithelial melanin unit in a nonspecific way. The ultrastructural morphology of melanocytes and keratinocytes was studied in clinically pigmented and nonpigmented gingival tissue from 11 smokers and 4 nonsmokers aged 15 to 50 years. No differences were found between clinically pigmented lesions in smokers and nonsmokers. In contrast, clinically nonpigmented tissue of smokers contained significantly more melanin-loaded keratinocytes compared with nonpigmented tissue from nonsmokers. In tissue of smokers and pigmented nonsmokers, melanocytes contained well melanized melanosomes (stages III and IV) compared with nonpigmented tissue of nonsmokers, where stage II melanosomes dominated. It is suggested that melanin in the oral mucosa plays a role as a binder of toxic products such as free radicals and polycyclic compounds. In this way, the epithelial melanin unit serves a protective function and prevents tissue damage. (Auth. Abs. Mod.)

84–1841. Holdstock, G.; Savage, D. A.; Wright, R. **Should Patients With Inflammatory Bowel Disease Smoke? (Letter).** *British Medical Journal* 288(6420): 862, March 17, 1984.

The original authors reply to a letter (*British Medical Journal* 288(6420): 862, March 17, 1984) criticizing their report (*British Medical Journal* 288(6414): 362, February 4, 1984) that suggested ulcerative colitis patients who smoked had more frequent and severe relapses. It is noted that the original study was designed to ascertain whether smoking was associated with severity of disease and not to determine if smoking was a causal or protective factor in ulcerative colitis or Crohn's disease. It is agreed that the ratio of the two types of inflammatory bowel disease is high, but diagnosis was made without knowledge of smoking habits, and probably reflects an increasing trend in Crohn's disease incidence. Also, the possibility of type 1 statistical error was recognized, and the Mann-Whitney test was used to check results. However, since 69 percent of patients with continuous symptoms were current smokers compared with only 20 percent of those with intermittent symptoms, the earlier statement that patients with inflammatory bowel disease should stop smoking is considered valid. See also 84-1184, 84-1845.

84–1842. Knott, V. J. **Electrodermal Activity During Aversive Stimulation: Sex Differences in Smokers and Non-Smokers.** *Addictive Behaviors* 9(2): 195-199, 1984.

Skin conductance activity was monitored in 16 male and 18 female smokers and 16 male and 16 female nonsmokers during high-intensity auditory stimuli. Frequency of response varied between groups, but not significantly: 33.3 percent of female smokers, 12.5 percent of male smokers, 12.5 percent of female nonsmokers, and 6.3 percent of male nonsmokers failed to show responses to any of the stimuli. Among responders, analysis of response amplitudes showed significantly higher responses among smokers, with responses from females significantly higher than those of males. Smokers also exhibited significantly higher mean resting levels, and levels for females were again significantly higher than in males. It is concluded that smokers are characterized by a state of relative hyperarousal and hypersensitivity to stimulus input, and it is suggested that smoking is a means, especially among females who smoke more in high-arousal situations, of "normalizing" physiological responsivity.

84–1843. Kurata, J. H.; Haile, B. M. **Epidemiology of Peptic Ulcer Disease.** *Clinics in Gastroenterology* 13(2): 289-307, May 1984.

Many studies have shown an association between smoking and peptic ulcer in males, with a twofold dose-dependent risk compared with nonsmokers. Ulcers heal more slowly, and smokers also have increased relapse rates. The association in females is not as strong. There are about 4 million active peptic ulcer cases in the United States; 350,000 new cases are diagnosed/year, with a 4:1 ratio of duodenal to gastric ulcers. Marked decreases in hospitalization (for duodenal) and mortality rates (for both) have occurred in recent years. In addition to smoking, environmental factors associated with peptic ulcers are regular aspirin use and prolonged use of steroids. Genetic factors play a role in both types. In most Western countries, morbidity from duodenal ulcer is more common than from gastric ulcer, but mortality rates are similar (about 3,000 deaths/year in the United States for each type). In Japan, both morbidity and mortality are higher for gastric ulcer than for duodenal ulcer.

84–1844. Lam, S.-K. **Pathogenesis and Pathophysiology of Duodenal Ulcer.** *Clinics in Gastroenterology* 13(2): 447-472, May 1984.

Duodenal ulcer research data indicate that there is a relationship between smoking and dyspeptic ulcer. Studies have reported 1.3 to 5.0 times higher incidence in smokers than in nonsmokers. It is likely that smoking increases basal acid output in some patients and also inhibits pancreatic bicarbonate secretion, although results from smoking and gastric acid secretion studies have been conflicting. Duodenal ulcer patients who smoke also show abnormally low levels of gastric mucosal histamine. Smoking's adverse effect on duodenal ulcer healing appears well established, and it is considered fair to conclude that smoking is a causal factor in some patients. The role of genetics and other epidemiological and environmental factors, such as stress and analgesics, is discussed. Clinical aspects of duodenal ulcer, including the time, role, and inhibition of gastric secretion, gastric motor function, and mucosal repair, are also covered.

84–1845. Langman, M.J.S.; Logan, R.F.A. **Should Patients With Inflammatory Bowel Disease Smoke? (Letter).** *British Medical Journal* 288(6420): 862, March 17, 1984.

A previous report (*British Medical Journal* 288(6414): 362, February 4, 1984) that found a low incidence of smoking among patients with ulcerative colitis is criticized for concluding that smokers have a reduced risk of the disease. It is claimed the conclusion is not justifiable because (1) the standard deviations quoted indicate data were not distributed normally; (2) in analyzing so many variables, "significant" differences could occur by chance; (3) the prevalence of the two types of inflammatory bowel disease is different from that usually found in the United Kingdom; and (4) no information about smoking habits prior to disease onset or data on controls are provided. Also, there was no evidence to support the claim that smoking leads to Crohn's disease rather than ulcerative colitis, although it is agreed that this may be likely. See also 84-1184.

84–1846. Lewis, D.; Capell, H. A.; McNeil, C. J.; Smith, W. E.; Brown, D. H. **Cigarette Smoking and the Clinical Outcome of Gold Therapy in Rheumatoid Arthritis. (Letter).** *Journal of Rheumatology* 11(1): 111-112, February 1984.

Results show that smoking affects erythrocyte gold levels with auranofin (AF) treatment and with sodium aurothiomalate (GSTM) treatment, but has no effect on the clinical outcome of chrysotherapy for rheumatoid arthritis. Previous studies (*Journal of Rheumatology* 9(4): 527-531, and 532-535, July-August 1982) had reported on differences in the distribution of gold in the blood of smoking vs. nonsmoking rheumatoid arthritis patients undergoing chrysotherapy. This prospective study randomly assigned patients to GSTM (n=30, 14 smokers), AF (n=30, 12 smokers), or AF placebo (n=30, 15 smokers) treatment. It is suggested that measurement of erythrocyte gold levels is of little value in predicting clinical response, since there was an equal distribution of smokers to nonsmokers among the improvers and nonimprovers in the three treatment groups. It may be that the study of the form of gold involved in transfer across cell membranes and their reactivities may well prove more useful in attempting to define the therapeutic effects of chrysotherapy. See also 83-0026, 83-0387.

84–1847. Matsuo, T.; Doi, K.; Amano, K. **Santen Hikakushiki Shutaiho ni Yoru Ewshukinodo Sokutei. [The Measurement of Odor Concentration of Cigarette Smoke by the Triangle Odor Bag Method.]** *Scientific Papers of the Central Research Institute, Japan Tobacco and Salt Public Corporation* 125: 33-39, 1983, Japanese.

No significant difference in mean perceived olfactory thresholds of cigarette smoke was observed in smokers (n=10) and nonsmokers (n=10). Individual threshold values were highly reproducible on successive tests and showed less fluctuation than among different panel members. Using the triangle odor bag method, immediate measurements were significantly higher than those taken after the bag had stood for an hour before testing. Odor concentrations of whole smoke did not differ from those of gas and vapor phase. No significant differences in odor concentrations were observed between smoke samples from five brands of manufactured cigarettes; four of the five brands produced less odor than smoke from unblended flue-cured cigarettes. (Auth. Abs. Mod.)

84–1848. McCloy, R. F.; Greenberg, G. R.; Baron, J. H. **Duodenal pH in Health and Duodenal Ulcer Disease: Effect of a Meal, Coca-Cola, Smoking, and Cimetidine.** *Gut* 25(4): 386-392, April 1984.

Smoking cigarettes did not affect intraluminal duodenal pH (recorded with a combined miniature electrode every 10 or 20 seconds for 5 hours) in an analysis of 11 duodenal ulcer patients and 8 control patients and controls. Seven patients and controls smoked 28 cigarettes, and duodenal pH readings were compared 30 minutes before and 30 minutes after starting a cigarette. After a meal, duodenal mean pH and log mean hydrogen ion activity (LMHa) fell significantly in both controls and patients with duodenal ulcer, with more periods of duodenal acidification and alkalinization in the duodenal ulcer group. An exogenous acid load (Coca-Cola) significantly increased periods of duodenal acidification and reduced alkalinization in both groups. Cimetidine significantly increased mean pH and LMHa and abolished the brief spikes of acidification in four of five patients with duodenal ulcer. Peak acid output (but not basal acid output) was significantly correlated with duodenal mean pH and LMHa, but not with periods of duodenal acidification. Smoking did not affect duodenal pH in either group. (Auth. Abs. Mod.)

84–1849. Norden, G.; Nyberg, G. **Smoking and Diabetic Nephropathy.** *Acta Medica Scandinavica* 215(3): 257-261, 1984.

Forty-seven patients with insulin-dependent diabetes (IDDM) and diabetic nephropathy and 47 controls with IDDM without diabetic nephropathy were interviewed about previous and current smoking habits. Patients were matched according to sex, age, age at onset, and duration of diabetes. All patients in the nephropathy group had proteinuria and decreased glomerular filtration. None in the control group had ever had proteinuria as tested by dipstick. The total amount of smoking until date of interview was estimated for each individual and presented as an index. Patients with nephropathy had a significantly higher smoking index than controls. In the nephropathy group there were also more current smokers, more heavy smokers, and fewer individuals who had never smoked than in the control group. The link between diabetic renal microangiopathy and smoking may be through mechanisms such as increased platelet aggregation, accentuated tissue hypoxia, and hemodynamic or metabolic effects of repeated noradrenaline release. (Auth. Abs.)

84–1850. Perry, R. J.; Griffiths, W.; Dextraze, P.; Solomon, R. J.; Trebbin, W. M. **Elevated Nicotine Levels in Patients Undergoing Hemodialysis. A Role in Cardiovascular Mortality and Morbidity?** *American Journal of Medicine* 76: 241-246, February 1984.

Data show markedly higher nicotine levels in hemodialysis patients ($n=10$, mean age 54 years) compared with controls ($n=10$, mean age 28 years) in an evaluation of the effects of tobacco smoking in these patients. Serum nicotine levels were assayed in patients with end-stage renal disease undergoing maintenance hemodialysis. Specimens were obtained before and after smoking one cigarette and following dialysis or an equivalent period in control subjects. Serum nicotine levels in control subjects measured 19.0±7.2 ng/ml initially, 36.1±8.2 ng/ml after smoking, and 9.3±3.5 ng/ml after a period of 4.35 hours. Values for hemodialysis patients were 76.6±16.8 ng/ml ($p<0.004$), 132.9±19.7 ng/ml ($p<0.001$), and 51.9±10.5 ng/ml ($p<0.001$), respectively. It is concluded that tobacco smoking may pose increased cardiovascular risks to patients with end-stage renal disease undergoing maintenance dialysis because of increased nicotine levels. (Auth. Abs. Mod.)

84–1851. Rees, T. D.; Liverett, D. M.; Guy, C. L. **The Effect of Cigarette Smoking on Skin-Flap Survival in the Face Lift Patient.** *Plastic and Reconstructive Surgery* 73(6): 911-915, June 1984.

Data demonstrate that a significant number of skin sloughs observed after face lift surgery are due to patients' cigarette smoking. Of 1,186 patients who underwent rhytidectomy, 121 had some degree of skin slough (unassociated with hematoma). Of the 91 patients for whom smoking histories were available, 73 (80 percent) admitted to smoking more than a pack of cigarettes/day at time of surgery. Thus a face lift patient who smokes has 12.46 times the chance of suffering skin slough than a patient who does not smoke. Approximately 74 percent of skin sloughs are due to smoking. The implications for surgeons, in terms of patient selection and preoperative counseling, are noted.

84–1852. Saville, P. D. **Postmenopausal Osteoporosis and Estrogens. Who Should Be Treated and Why.** *Postgraduate Medicine* 75(2): 135-138, 142, February 1, 1984.

Women who smoke are more at risk of osteoporosis than women who do not smoke. Others at increased risk include light-skinned races; small, lightweight women; women who have early menopause; those with rheumatoid arthritis or undergoing corticosteroid therapy; and women with sedentary habits. Estrogen deficiency after menopause results in loss of skeletal calcium and increased risk of bone fractures. Administration to postmenopausal women of a daily dose of 0.625 mg of conjugated equine estrogen, 1 mg estradiol-17B, or 0.15 mg ethinyl estradiol prevents changes. However, the safety of long-term estrogen administration has not been established by large-scale controlled studies; therefore, hormone treatment should be reserved for patients with symptoms of estrogen deficiency or for the subsets at increased risk of osteoporotic bone fractures. (Auth. Abs. Mod.)

84–1853. Shousha, S.; Barrison, I. G.; El-Sayeed, W.; Khan, S.; Parkins, R. A. **A Study of Incidence and Relationship of Intestinal Metaplasia of Gastric Antrum and Gastric Metaplasia of Duodenum in Patients With Nonulcer Dyspepsia.** *Digestive Diseases and Sciences* 29(4): 311-316, April 1984.

Intestinal metaplasia was significantly higher in patients who smoked ≥ 10 cigarettes/day ($p<0.002$) in a study of endoscopic biopsies from 120 patients (67 males, 53 females) with nonulcer dyspepsia. Gastric metaplasia was not related to smoking. Intestinal metaplasia was found in 29 percent of antral biopsies and gastric metaplasia in 39 percent of duodenal biopsies; 9 percent had both. Gastric metaplasia was associated with duodenitis, and incidence was significantly higher in males ($p<0.001$) and in patients with a history of high or moderate alcohol intake ($p<0.02$). Findings suggest that duodenitis and duodenal ulcers may represent different facets of a single disease spectrum. (Auth. Abs. Mod.)

84–1854. Thomas, J. M.; Misiewicz, G. **Histamine H_2-Receptor Antagonists in the Short- and Long-Term Treatment of Duodenal Ulcer.** *Clinics in Gastroenterology* 13(2): 501-541, May 1984.

Results from studies of the effect of smoking on healing of peptic ulcer are conflicting; some report that smoking retards peptic ulcer healing and increases pepsin I production, six report that smoking slows duodenal ulcer healing, while four found that it does not. Two report that smoking stimulates gastric acid secretion and two that it does not affect inhibition of pentagastrin-stimulated acid secretion. Cimetidine and ranitidine, the two clinically available H_2 antagonists for the treatment of duodenal ulcers, are discussed in terms of short-term treatment (smoking, clinical trials, comparisons with each other and with other drugs, and alcohol effect) and also long-term maintenance treatment. Among long-term considerations are intermittent treatment, demand treatment schedules, medical vs. surgical management, and unwanted effects, including inhibition of drug metabolism and drug interactions, hyperprolactinemia and antiandrogenic activity, bone marrow effects, cardiovascular effects, and potential carcinogenicity.

84–1855. Wald, N.; Ritchie, C. **Validation of Studies on Lung Cancer in Non-Smokers Married to Smokers. (Letter).** *Lancet* 1(8385): 1067, May 12, 1984.

Assessment of passive smoke exposure in men married to smokers adds plausibility to findings of excess lung cancer risk among nonsmokers with spouses who smoke. Among 121 nonsmoking married men, 20 were married to smokers and had mean urinary cotinine concentrations of 25.2 ng/ml, significantly higher than the mean of 8.5 ng/ml found in the 101 men with nonsmoking wives. Also, men married to smokers were more tolerant of other people's smoke; thus, duration of smoke exposure outside the home was greater (mean 16.4 hours/week) than among those married to nonsmokers (mean 10.0 hours/week). Mean total exposures/week were 23.2 and 11.0 hours, respectively. Although a spouse's smoking accounts for only a part of daily passive tobacco smoke exposure, nonsmokers married to smokers were nonetheless more generally exposed to smoke than those married to nonsmokers, and marriage to a smoker was a good indicator of high passive smoke exposure.

See also, **84–1814, 84–1815**

BEHAVIORAL AND PSYCHOLOGICAL ASPECTS

84–1856. Afifi, L. A. **An Exploratory Study to Investigate the Determinants of Health Behavior Towards Smoking in Bahreini Secondary School Males.** Doctoral Dissertation, University of Iowa, Graduate College of Education, Iowa City, University Microfilms International 83-25128, 1984, 193 pp.

In an effort to ascertain whether the Health Behavior Model of social learning theory could be used to measure health behavior in an Arab population and to investigate determinants of health behavior toward smoking in Arab adolescents, a pilot research project was undertaken among 649 randomly selected male secondary school students in Bahrein. The test design included social structure variables, stated smoking behavior, health beliefs concerning smoking, health locus of control, and health valuing. Students (aged 14 to 21) were tested before and after an educational program (18-min videotape). Among the participants, 82.1 percent reported not smoking, 11.0 percent consumed <5 cigarettes/day, and 3.8 percent smoked >5 cigarettes/day (3.1 percent did not answer). At pretest, smokers reported a significantly lower perceived susceptibility to smoking-related health problems than nonsmokers. Smokers also claimed fewer cues to smoke, but no differences in health locus of control were found. Smokers reported fewer extended family members at home, fewer close paternal relatives living next door, and less perceived parental objection to smoking. For smokers, referral on health matters was more often to friends and brothers, while nonsmokers referred more frequently to their fathers. A generally high value on health was reported, with no differences between smokers and nonsmokers. Following the videotape, nonsmokers showed an increased perception in both seriousness and susceptibility to smoking-related health problems, while smokers showed only an increase in the perceived seriousness of the problems. It is concluded that social learning theory appears to be feasible for use in health behavior determination in the Bahreini adolescent, although it cannot be assumed that health behavior or determinants are identical in all Arab countries.

84–1857. Akers, R. L.; Massey, J.; Clarke, W.; Lauer, R. M. **Are Self-Reports of Adolescent Deviance Valid? Biochemical Measures, Randomized Response, and the Bogus Pipeline in Smoking Behavior.** *Social Forces* 62(1): 234-251, September 1983.

Findings of two confidential questionnaire surveys of students in grades 7 through 12 (2,194 in first year, 2,150 in second) suggest that respondents felt reasonably secure and satisfied that answers would remain confidential and tended to report truthfully under all conditions of data collection. Responses to the questionnaire were validated by responses to an anonymous "randomized response" instrument and a biochemical measure of smoking (salivary thiocyanate). Both measures support the validity of questionnaire responses. The randomized response measure is an anonymous self-report check on the accuracy of confidential self-reports, and the biochemical measure provides a strong validity measure that is not subject to the problems of deliberate faking. It is suggested that in studies where proper conditions are provided and there is reason to believe that respondents accept assurances of confidentiality, but where collection of saliva samples is not feasible, sampling can be omitted without much risk of loss of response validity. Validity of responses was not significantly affected by the introduction of a "bogus pipeline" condition. (Auth. Abs. Mod.)

84–1858. Alexy, B. B. **Goal-Setting and Health Risk Reduction.** Doctoral Dissertation, University of Pennsylvania, School of Nursing, Philadelphia, University Microfilms International 83-20851, 1983, 86 pp.

To determine whether collaboration between a health care provider (nurse) and a client would result in greater health risk reduction than if the plan was formulated entirely by the provider, 152 healthy subjects from three industries (insurance, utility company, and food production), aged 27 to 66 years, completed the Personal Risk Profile Questionnaire. The 52 subjects (38 males, 14 females) in the provider goal-setting group were given profile results and instructed, as applicable, to stop smoking, lose weight, increase physical activity, reduce alcohol consumption, use seat belts, and among women, to perform breast self-examination monthly. For the 40 men and 14 women in the collaborative goal-setting group, health goals were negotiated between the subject and the provider. Controls (26 males, 20 females) received feedback from the profile but were not involved in goal-setting. At 3-month followup, no significant changes in smoking behavior occurred in any group. Overall, those with preset goals and a greater number of goals were more successful in reducing health risks. No contact was made with participants during the 3-month interval, and it may be necessary to provide periodic psychological support to encourage behavioral change. Also, the higher level of success with the provider goal-setting group may indicate that individuals prefer structured programs. Although quitting smoking would have conferred a greater benefit, exercise and weight reduction were more frequently chosen as risk reduction goals.

84–1859. Baugh, C. L. **Prediction of Smoking Behavior Using the HBM, Health Locus of Control and Self-Esteem.** Doctoral Dissertation, Indiana University, School of Nursing, Bloomington, Indiana, University Microfilms International 84-00846, May 1983. 100 pp.

Although the expanded Heath Belief Model (HBM) did predict ex-smokers and smokers, the addition of locus of control and self-esteem as constructs did not substantially increase the predictive power of the HBM at a significant level. Smokers ($n=109$) of at least a pack/day and ex-smokers ($n=103$) were asked to respond to tools measuring the original HBM variables, smoking-related health locus of control variables, and self-esteem. Factors used as dependent variables were daily consumption of cigarettes and years smoked, and in ex-smokers, the number of years since quitting. All of the hypotheses were rejected because the independent variables were unable to account for substantial variance in the prediction of smoking behavior as measured by the dependent variables. The HBM variables, however, did enter the multiple regression equation at a significant level ($p<0.05$) with at least one dependent variable. Susceptibility accounted for the most variance on all dependent variables among ex-smokers. Benefits of not smoking was the only variable that accounted for variance at a significant level for smokers on both dependent variables. Different variables entered the multiple regression equation to predict smokers and ex-smokers; thus, the two behaviors did not prove to be simply antipodal. (Auth. Abs. Mod.)

84–1860. Bauman, K. E.; Fisher, L. A.; Bryan, E. S.; Chenoweth, R. L. **Antecedents, Subjective Expected Utility, and Behavior: A Panel Study of Adolescent Cigarette Smoking.** *Addictive Behaviors* 9(2): 121-136, 1984.

Research to determine whether (1) subjective expected utility (SEU) influences behavior, (2) behavior influences SEU, (3) one direction of the SEU-behavior relationship is stronger than the other, and (4) SEU explains why general social and psychological variables are related to behavior, particularly cigarette smoking by adolescents, is summarized. In a panel study of 1,334 adolescents who completed two questionnaires about a year apart, SEU was found to be related to smoking behavior. This relationship reflected both the influence of SEU on behavior and the influence of behavior on SEU. Although a dominant direction of the relationship could not be identified, among those who began the study as nonsmokers, SEU explained some but not all of the relationship between antecedent variables and smoking behavior, and SEU compared favorably with other variables as a predictor of behavior. It is concluded that SEU should be considered when explaining behavior, and in particular, that SEU contributes to the understanding of cigarette smoking by adolescents. (Auth. Abs. Mod.)

84–1861. Brod, M.; Hall, S. M. **Joiners and Non-Joiners in Smoking Treatment: A Comparison of Psychosocial Variables.** *Addictive Behaviors* 9(2): 217-221, 1984.

Data from a study of 108 smokers to differentiate smoking cessation treatment joiners from nonjoiners found that nonjoiners had a lower self-efficacy expectation than treatment joiners. Self-efficacy is the person's belief that he or she can successfully carry out a behavior called for by a particular situation to produce a desired outcome, based on past performance accomplishments, vicarious experiences, verbal persuasions from others related to the behavior change, and cues from internal physiological states used to assess emotional and/or physical states. Other variables studied were demographic characteristics, tobacco and drug use, anxiety, and health locus of control. Nonjoiners were also found to be more anxious and less educated than joiners. It is concluded that self-efficacy, which correlated significantly with anxiety, is related to joining treatment behavior. The measure of self-efficacy used and the dimensions of the concept that are being tapped should be considered when interpreting any results. The differences in education noted are probably unique to a university treatment setting. (Auth. Abs. Mod.)

84–1862. Chait, L. D.; Griffiths, R. R. **Effects of Methadone on Human Cigarette Smoking and Subjective Ratings.** *Journal of Pharmacology and Experimental Therapeutics* 229(3): 636-640, June 1984.

Results demonstrate that methadone can produce substantial increases in cigarette smoking and may have implications regarding the proposed role of endogenous opioids in the smoking process. The effects of varying methadone dosage on the smoking behavior of five male methadone-maintenance patients were examined. Isolated subjects smoked their regular brand of cigarettes ad libitum in a naturalistic laboratory environment while reading or watching television. Ninety minutes before each daily 2-hour smoking session, subjects received either the placebo dextromethorphan (a taste blind) or one of three doses of methadone at 0.5, 1.0, or 2.0 times the regular maintenance dose (40-60 mg). Every subject received each treatment five times, in a mixed order across days. Methadone pretreatment resulted in a dose-related increase in number of cigarettes smoked/session (from a mean of 2.8 after placebo to 5.6 after the high dose of methadone). Total time spent puffing increased from a mean of 27 sec after placebo to 74 sec after high methadone dose. Carbon monoxide levels in expired air showed corresponding dose-related increases. Methadone administration also resulted in dose-related decreases in pupil diameter and increases in subjective ratings of smoking satisfaction and dose strength. Dextromethorphan had no significant effects on any measure of smoking behavior or subjective response. (Auth. Abs. Mod.)

84–1863. Charlton, A. **The Brigantia Smoking Survey: A General Review.** In: Hobbs, P. (Editor). *Public Education About Cancer. Recent Research and Current Programmes.* UICC Technical Report Series, Volume 77. Geneva, 1984, pp. 92-102.

The Brigantia questionnaire survey sampled 20,000 students aged 9 to 19 years in urban and rural areas of Cumbria, Tyne, and Wear counties in northern England. A regular smoker was defined as consuming ≥ 1 cigarettes/week; 0.3 percent of all 9-year-olds and up to 28 percent of male and 36 percent of female 19-year-olds were regular smokers. Occasional smokers (< 1 cigarette/week) never exceeded 9 percent of any age group, although 40 percent of the 17- to 18-year-olds had tried but had not continued smoking. It appears that those who do not experiment before age 16 probably never will. Only 7 percent of all smokers, but 42 percent of heavy smokers, intended to continue cigarette use. Both smoking habits and perceived attitudes toward the subject's smoking behavior among parents, siblings, teachers, and friends showed significant influence on the students' smoking habits. Knowledge about the health effects of smoking was comparable between smokers and nonsmokers, and all agreed that smoking "makes you smelly." Nonsmokers thought that people smoked to show off, while smokers cited calming the nerves, weight loss, and feeling confident as reasons for smoking. Almost 75 percent of regular smokers under 16 years of age buy cigarettes from shops, where cigarettes cannot legally be sold to anyone under age 16. Findings indicate that young people's opinions on smoking, if not their smoking habits, have changed considerably since 1966, and the new information will be used to plan and evaluate new strategies for prevention.

84–1864. Christen, A. G.; Swanson, B. Z. **Orally Used Smokeless Tobacco as Advertised in the Metamorphic Trade Cards of 1870-1900.** *Bulletin of the History of Dentistry* 31(2): 82-86, October 1983.

Since the early 1970s, American males have dramatically increased consumption of smokeless tobacco, which produces significant dental changes, including bad breath, discolored teeth and fillings, tooth abrasion, decreased acuity of taste and smell, gingival recession, advanced periodontal destruction, leukoedema, soft tissue erythema, leukoplakia, and cancer. Both past and present advertising emphasize that smokeless tobacco satisfaction is primarily reserved for males who enjoy the camaraderie of using the product and who appreciate full-flavored tobacco taste. Clever metamorphic trade cards of the late 1800s are illustrated. Modern advertising features popular sports personalities and entertainers who endorse the products, and implies that smokeless tobacco is innocuous.

84–1865. Cohen, D. R. **Utility Model of Preventive Behaviour.** *Journal of Epidemiology and Community Health* 38(1): 61-65, March 1984.

As an alternative to psychosociological analysis, the utility model of preventive behavior is presented, including smoking as an example. According to the model, all preventive actions are viewed as the consumption of those "goods" that affect the risk of illness or injury. Levels of consumption depend on, among other things, utility (perceived benefit) and cost. Total utility yielded by risk-affecting goods is the sum of that derived from the use value of the goods, if any, and that from reduced anxiety resulting from reduced risk. Many risk-affecting goods are consumed mainly for their use value; e.g., cigarette smoking is a risk-increasing good that provides both positive and negative utility to smokers. For most smokers, the positive utility from smoking overshadows the negative. It is suggested that an antismoking campaign aimed at reducing perceptions of positive utility, rather than increasing perceptions of disutility, may prove a more efficient way of reducing smoking. (Auth. Abs. Mod.)

84–1866. Coppotelli, H. A. **Spouse Support in Smoking Cessation by Women.** Doctoral Dissertation, Duke University, Department of Psychology, University Microfilms International 83-25662, 1983, 166 pp.

Women's perceptions of spouses as facilitating, in general and with regard to quitting smoking, were significantly related to cessation efforts. Analyses suggested that this influence occurred primarily through the facilitating spouse's fostering a positive interpersonal environment and alleviating negative emotional conditions and/or feelings of drudgery associated with relapse. Spousal influence was analyzed in 125 women quitting smoking, who completed questionnaires for demographic and smoking habit data and their perceptions and experience when they first quit and again at 6- to 8-week followup. Women rated spouses' contributions in a number of critical areas and their views of themselves in analogous items. Factor analysis with oblique rotation revealed three common factors in initial spouse perceptions: facilitation, confidence, and nondomination. Three analogous factors in initial self-perceptions were experienced support, personal efficacy, and quit control. The spouse factor structure was more interrelated and emphasized facilitation, with 74 percent of the common variance, and demonstrated that perceptions of the spouse were a composite of general and cessation-specific attributions.

Facilitation was the primary predictor of cessation outcome (32 percent of variance). Secondary predictors were the spouse's being an ex-smoker, the subject having a professional career, and the subject formerly smoking for ritual or habitual reasons. Use of criteria in a subsequent discriminant analysis resulted in correct classification of 80 percent of test cases. (Auth. Abs. Mod.)

84–1867. D'Hondt, W.; Vandewiele, M. **Attitudes of Senegalese Schoolgoing Adolescents Towards Tobacco Smoking.** *Journal of Youth and Adolescence* 12(4): 333-353, August 1983.

In Senegal, as in most developing countries, there is a close relationship between smoking and growing adoption of Western values and ways of life concomitant with industrialization and urbanization, which has created new needs and further maladjustments for which smoking has appeared to be a miraculous panacea. Also, smoking has increased because of advertising campaigns by tobacco interests. Many Senegalese adolescents have focused on cigarette smoking as a palliative for the stress and insecurity that they feel in their society, finding it also a "rite of passage" for their initiation into their age group and into self-confidence. Survey data (1980-1981) showed that 47.3 percent of 731 students aged 17 to 21 were current smokers. However, several factors, more psychological and cultural than economic, militate against smoking. Many adolescents are aware that smoking is hazardous to their health although precise information is often lacking. As students grow into adulthood and greater self-confidence and become more responsive to notions of national identity and cultural authenticity, they tend to outgrow the smoking mania. Thus, in a young developing nation such as Senegal, where the youth are still open to the virtues of scientific discoveries and to the genuine values of the cultural past, where religious authority and the power of government are still strong, a well-conceived and carefully implemented antismoking strategy should yield fruitful results. (Auth. Abs. Mod.)

84–1868. Epstein, L.; Tamir, A. **Health-Related Behavior of Adolescents: Change Over Time.** *Journal of Adolescent Health Care* 5(2): 91-95, April 1984.

Repeat interviews with 87 male and 94 female Israeli high school students (at age 16 and again 2 years later) found that smoking, use of drugs, sexual behavior, and alcohol consumption at the age of 16 was related to dropout from school. At age 16, 15.4 percent of males and 8.7 percent females were smokers. By age 18, 36.1 percent and 31.3 percent, respectively, were smokers. The longitudinal study was carried out in a large, comprehensive secondary school. Data indicate that more females started smoking between the ages of 16 and 18, but a higher proportion of girls stop within a short period. The numbers of adolescents beginning to smoke indicates a failure of preventive education. The correlation of behaviors suggests that it may be possible to construct a risk profile for the developing adolescent (including age, cigarette smoking, sexual activity, and use of hashish and alcohol) that might identify groups of adolescents who are developing behaviors that would place their health at risk or lead to school dropout. The profile might allow intervention aimed at changing behaviors.

84–1869. Field, M. D. **Strong Sentiment to Restrict Smoking in Public Places.** California Poll, Release No. 1237, San Francisco, Field Institute, February 29, 1984, 5 pp.

A survey of opinions on smoking in public places found that ex-smokers now outnumber smokers in California: 26 percent of the sample were current smokers, 35 percent ex-smokers, and 39 percent never smokers. In restaurants, 77 percent of respondents wanted smoking restrictions (special areas only) and 16 percent opted for outright prohibition, while in workplaces, 70 percent favored restrictions and 16 percent preferred a ban. Concerning public transportation, 59 percent favored restrictions on planes and 50 percent wanted restrictions on buses and trains; 38 and 46 percent, respectively, wanted total smoking bans on planes and buses/trains. A majority (58 percent) also favored special smoking areas in motels and hotels, while 33 percent opted for no restrictions. Most (86 percent) ex-smokers and nonsmokers, but only 50 percent of current smokers, believe smoking is very hazardous to health: 54 percent vs. 17 percent, respectively, believe passive smoking is hazardous. By a 58 to 39 percent margin, current smokers agree that smokers should refrain from smoking in the presence of nonsmokers. While the majority of ex-smoker and nonsmokers are bothered by cigarette smoke, the majority of smokers complained only about cigar smoke.

84–1870. Fouriaud, C.; Jacquinet-Salord, M. C.; Degoulet, P.; Aime, F.; Lang, T.; Laprugne, J.; Main, J.; Oeconomos, J.; Phalente, J.; Prades, A. **Influence of Socioprofessional Conditions on Blood Pressure Levels and Hypertension Control.** *American Journal of Epidemiology* 120(1): 72-86, July 1984.

Cigarette smoking and alcohol consumption (known to be linked with blood pressure) were more frequent in the lowest occupational categories and might explain the differences in blood pressure observed in an investigation of the influence of socioeconomic and professional factors on the prevalence and treatment of arterial hypertension. Subjects (3,896 men and 2,769 women) were employed by small- and medium-sized companies in the Paris area. Mean systolic blood pressure (SBP) was significantly higher in the lowest occupational categories than in the higher categories even after adjustment for age and body mass index. Diastolic blood pressure (DBP) differed among categories, and differences were still significant after adjusting for age, but not for body mass index, suggesting that body mass may be partly responsible for the differences in DBP. Prevalence of hypertension (14.7 percent) was negatively associated with high occupational category ($p<0.001$). Multivariate analysis confirmed the influence of occupational category on SBP ($p<0.001$). Both the percentage of treated patients and compliance with treatment were positively associated with occupational category ($p<0.01$ in both). Among women, SBP of treated hypertensives was higher in the lower compared with the higher occupational categories ($p<0.05$). It is suggested that the differences in blood pressure levels observed are partly due to working conditions, and that poor compliance with treatment among the low occupational categories enhances the differences. (Auth. Abs. Mod.)

84–1871. Hackbarth, D. P. **A Process Model of Smoking and Quitting Behaviors.** Doctoral Dissertation, University of Illinois at Chicago, University Microfilms International 84-04403, 1984, 304 pp.

An integrated process model was developed using the theory of reasoned action, which postulates that attitudes, beliefs, and subjective norms predict intentions and intentions predict behaviors, to show that quitting smoking is a process rather than an isolated event. Questionnaire responses from 423 subjects (120 men, 303 women; 35 percent smokers, 24 percent ex-smokers) provided demographic characteristics, self-reported smoking habits, and past smoking and quitting history; scales measured attitudes, beliefs, and norms concerning smoking, quitting, health effects, and attitudes of significant others. Discriminant analysis was used to select the most important attitude and subject norm constructs that discriminated between smokers, ex-smokers, and nonsmokers at one point in time. Similar analysis was used to discriminate among smokers who intended to smoke, cut down, or quit during the 6-month study. Data revealed that (1) clusters of attitudes and subjective norms could accurately differentiate smokers, ex-smokers, and nonsmokers in both sexes; (2) a monotonic relationship existed (smokers scored at one end of a continuum, ex-smokers scored in the middle, and nonsmokers at the opposite end); (3) among women, intentions to smoke, cut down, or quit were significantly related to quitting attempts, but not to reduction in consumption or cessation; and (4) most successful ex-smokers of both sexes reported having made several attempts to quit prior to final cessation. It is concluded that attitudes and subjective norms change from smoker-like to nonsmoker-like during a quitting process, suggesting that smoking cessation is a process with identifiable and sequential attitude and subjective norm stages. (Auth. Abs. Mod.)

84–1872. Halikas, J. A.; Weller, R. A.; Morse, C. L.; Hoffmann, R. G. **Use of Marijuana and Other Drugs Among Adult Marijuana Users: A Longitudinal Study.** *Comprehensive Psychiatry* 25(1): 63-70, January-February 1984.

Use of cigarettes, hallucinogens, stimulants, barbiturates, and opiates had declined in 97 adult regular marihuana users interviewed in 1970 and again in 1976. The number of persons smoking less than a pack/day had decreased; however, the number of heavier smokers had remained the same, and smoking behavior reported at original interview was highly correlated with smoking at followup. At first interview, the majority were using marihuana one to four times/week; by followup, most had either decreased to only occasional use or had increased to near daily use. Alcohol was more frequently used at followup. Five levels of drug use were examined: from those using very little alcohol and no cigarettes (level 1) to marihuana use (level 3), to use of substances from among four categories of "harder" drugs (level 5). The majority had moved to a lower level by followup, with only 10 percent moving to a higher level. Marihuana use frequency was related to extent of other drug use at the original but not at followup interview. Cessation of harder drug use was found to be predicted best by marriage and by having friends who were not marihuana users. (Auth. Abs. Mod.)

84–1873. Hanks, D. T. **Physician Modeling Influences on Patient Smoking.** Doctoral Dissertation, North Texas State University, Denton, University Microfilms International 84-04317, 1984, 184 pp.

Results did not confirm the hypothesis that older light and heavy smokers would differentially respond to a model's smoking or nonsmoking in a manner similar to younger light and heavy smokers. Heavy smokers ($n=27$, >20 cigarettes/day) smoked at significantly higher rates on all cigarette measures than light smokers ($n=27$, ≤ 19 cigarettes/day), and rates did not vary significantly relative to the presence or absence of model smoking. Results from exposure to a nonsmoking physician model who advised quitting, a smoking physician model who advised quitting, and a smoking physician model who did not comment on smoking relative to patient behavior and attitudes showed that patients smoked significantly more when the model advised quitting and significantly less when no comment was made, regardless of model smoking or nonsmoking. When data were reexamined according to locus of control and social desirability, it was found that, relative to other treatment conditions, internal patients smoked significantly more when advised to quit, while external patients smoked significantly more when the model smoked and significantly less when the model did not smoke. Low social desirability patients exhibited rates similar to the internal patients, and high social desirability patients smoked significantly more when the model was contradictory and significantly less when the model was congruent, relative to other modeling conditions. Thus, light—heavy distinctions were not considered as useful as locus of control and social desirability in predicting and understanding the interactive behavior of a model and observer. (Auth. Abs. Mod.)

84–1874. Kandel, D. **Substance Abuse by Adolescents in Israel and France: A Cross-Cultural Perspective.** *Public Health Reports* 99(3): 277-283, May-June 1984.

With respect to cigarettes, all alcoholic beverages, and illegal drugs, French youth consistently reported earlier age of onset, greater lifetime use, greater current use, more extensive involvement, and fewer sex differences than Israeli youth. Survey data for 609 Israeli and 499 French urban adolescents, aged 14 to 18 years, showed that cigarettes and alcohol were used more frequently than other illicit drugs; use of cigarettes and alcohol preceded use of illicit drugs; and marihuana was the most frequently used illicit drug. For all drugs surveyed, use in France exceeded that in Israel. More French youth had smoked than had drunk an alcoholic beverage other than cider, but the reverse was true in Israel. In France, 82 percent had tried cigarettes and 64 percent had smoked in the past 30 days; in Israel, 44 percent had tried cigarettes and 16 percent had smoked in the last 30 days. Of current smokers, 7 percent of Israeli vs. 16 percent of French youth consumed a pack or more/day, 32 vs. 46 percent less than a pack/day, and 61 vs. 38 percent smoked less than one cigarette/day.

84–1875. Knott, V. J. **Noise and Task Induced Distraction Effects on Information Processing: Sex Differences in Smokers and Non-Smokers.** *Addictive Behaviors* 9(1): 79-84, 1984.

Choice reaction time performance was monitored in 16 male and 16 female smokers and 16 male and 16 female nonsmokers during the separate and combined presentation of noise- and task-induced distraction. Behavioral measures of decision time and movement time were examined together with response errors. Female smokers exhibited significantly slower decision times compared with female nonsmokers during both separate and combined presentation of distractors. In addition, female smokers showed significantly longer decision times than male smokers during task-induced distraction. Significant differences were also observed with response errors, with task and combined noise- and task-induced distraction resulting in greater impairment in female smokers. Results indicate a relative central information processing deficit in female smokers. Discussion is focused on the role of attentional processes. (Auth. Abs.)

84–1876. Lewis, C. E.; Lewis, M. A. **Peer Pressure and Risk-Taking Behaviors in Children.** *American Journal of Public Health* 74(6): 580-584, June 1984.

A survey of 771 children in grades 5 through 8 found that students are presented with dares and challenges to place themselves at risk at an age before they are capable of understanding the causal relationship between an act, such as smoking, and the nonreversibility of its consequences. Peer pressure is defined as both part of the normal process of psychological development (dares and challenges stimulate actions that affirm affiliations and help establish identity and clarify values) and a major contributor to development of risk-taking behaviors. Students were asked about dares and challenges received from other children, and categories were established for classifying proposed actions, techniques utilized, and responses. Peer pressure was reported most frequently by eighth grade students. Smoking cigarettes was the major contributor to the substance abuse category among fifth graders, while smoking marihuana accounted for 50 percent of this category among seventh and eighth graders. About 50 percent of all dares encouraged problem behaviors that placed the children (or others) at risk for personal injury or the potential development of habits hazardous to their health. With increasing age, more occurred at school and fewer involved risk of personal injury. Among seventh and eighth graders, more boys were dared to perform acts of violence and more girls were challenged to be sexually active. (Auth. Abs. Mod.)

84–1877. Public Health Reports. **Survey Shows Continued Drop in Use of Most Drugs by High School Seniors.** *Public Health Reports* 99(3): 324, May-June 1984.

Data for 16,300 seniors in public and private schools show that smoking and use of heroin, cocaine, and other opiates remained unchanged, while there was a small increase in the use of PCP. However, the proportion of daily marihuana users declined for the fifth straight year (10.7 percent in 1978 to 5.5 percent in 1983). Use of amphetamines, barbiturates, methaqualone, and LSD has also declined. There was no evidence that alcohol was being substituted for other drugs; the percentage of daily alcohol users actually declined slightly (5.7 to 5.5 percent). Despite the steady overall decline, large subgroups remain whose drug use patterns are becoming heavier and more damaging; emergency room mentions for heroin and cocaine use increased steadily between 1980 and 1983.

84–1878. Shiffman, S. M.; Jarvik, M. E. **Cigarette Smoking, Physiological Arousal, and Emotional Response: Nesbitt's Paradox Re-Examined.** *Addictive Behaviors* 9(1): 95-98, 1984.

Because nicotine's paradoxical effects on arousal and mood are poorly understood, an attempt was made to replicate Nesbitt's Paradox (that smokers show less anxiety as smoking-induced physiological arousal increases). In smoking and sham-smoking sessions, 10 smokers (2 male, 8 female; average consumption ≥20 cigarettes/day for a year) were administered a series of progressively stronger electric shocks. Pain thresholds and Multiple Affect Adjective Checklist (MAACL) anxiety scores measured emotional arousal, while heart rate increases served as indices of physiological arousal. All measurements were taken automatically to minimize experimenter—subject interaction. Smoking increased heart rate, but had no effect on thresholds or MAACL scores. Emotional and physiological arousal showed a significant positive relationship (r=.65), contradicting Nesbitt's report of an inverse relationship, and suggesting that Nesbitt's Paradox may not be a robust phenomenon. (Auth. Abs. Mod.)

84–1879. Wells, K. B.; Ware, J. E., Jr.; Lewis, C. E. **Physicians' Practices in Counseling Patients About Health Habits.** *Medical Care* 22(3): 240-246, March 1984.

A model of two dimensions of physicians' behavior in counseling patients about four health habits (smoking, weight control, alcohol consumption, and regular exercise) was developed. The two dimensions are (1) indications for routine counseling and (2) aggressiveness of counseling style. To test the dimensions, a questionnaire was administered to a random sample of members of a Western County Medical Society in 1978 (response rate 76 percent) and Indications and Aggressiveness subscales were developed for each habit. Reliability and discriminant validity for all subscales was excellent. Descriptive results were consistent with expectations: e.g., physicians counseled more about smoking and weight control than about alcohol and exercise. Data show 52.4 percent counseled all smoking patients to quit, 17.5 percent advised heart and lung disease patients to stop smoking, 15.9 percent counseled only lung disease patients to quit, and 14.3 percent did not address patients' smoking habits. Of those who advised patients to quit smoking, only 48.5 percent initiated counseling, spent more than 2 minutes for counseling, and repeated advice more than once/year. (Auth. Abs. Mod.)

84–1880. Wigle, D. T.; Morgan, P. P. **The Tobacco Industry: Still Resourceful in Recruiting Smokers.** *Canadian Medical Association Journal* 130(12): 1537-1539, June 15, 1984.

The Canadian Ski Association (CSA) recently announced that the RJR-MacDonald Tobacco Company, a subsidiary of R. J. Reynolds, would sponsor several events in the CSA program through April 1988. The Canadian Medical Association protested the agreement in a letter to the CSA. CSA argued that the relationship with RJR-MacDonald was dictated by financial necessity. However, the CSA receives a large subsidy from the Department of National Health and Welfare for the purpose of promoting interest in sports and health among youth. Voluntary advertising restrictions specify that tobacco promotion should not be targeted at persons under 18 years of age; however, not only does the CSA have many participants under age 18, but the contract allows RJR-MacDonald the right to veto sponsorship of other products of interest to people in this age group. Tobacco industry apologists argue that advertising serves only to influence brand selection and does not recruit new smokers. Yet, 5 years after institution of an advertising ban in Norway, smoking rates were declining in both sexes. Excerpts from tobacco industry reports outlining current advertising strategy are included.

84–1881. Williams, D. G.; Tata, P. R.; Miskella, J. **Different "Types" of Cigarette Smokers Received Similar Effects From Smoking.** *Addictive Behaviors* 9(2): 207-210, 1984.

Forty-eight smokers (>10 cigarettes/day), aged 18 to 25 years, were divided into high-arousal and low-arousal situation smokers using the Situational Smoking Questionnaire. After abstaining from smoking between midnight and 10:00 a.m., subjects took letter cancellation tests (a low-arousal situation) while smoking cigarettes containing 1.3 mg nicotine (males) or 1.5 mg nicotine (females). Pulse rates were also observed. All subjects increased speed of performance with practice and smoking. However, although high-arousal smokers performed faster on letter cancellation tests, groups did not differ in extent of improvement. Also, there were no significant differences between groups in pulse rate changes.

See also, **84–1658, 84–1709, 84–1726, 84–1842, 84–1896**

SMOKING PREVENTION AND INTERVENTION

84–1882. AMA Council on Scientific Affairs. **Nonsmoking in Hospitals.** *Connecticut Medicine* 48(5): 297-305, May 1984.

Recommendations for guidelines to control smoking in hospitals are presented, in response to Substitute Resolution 146 (A-81), to the American Medical Association (AMA) House of Delegates. The guidelines, in part, specify that (1) physicians should take a leadership role in developing hospital smoking policies; (2) bedridden patients should be forbidden to smoke; (3) hospitals should guarantee smoke-free rooms upon request; (4) no tobacco products should be sold on hospital grounds; (5) hospitals should make smoking cessation programs available to patients, employees, and the community; and (6) smoking should be prohibited in certain areas and restricted to specifically designed, well-ventilated places. In developing the guidelines, the council explored and reviewed the effects of smoke on the nonsmoker, the fire hazards from smoking, public attitudes toward restrictions on smoking in public places, current hospital policies, and relevant other AMA policy. Obstacles to efforts to institute smoking control programs include concern for the rights of individual smokers, the costs of implementation, difficulty in enforcement, physician and staff attitudes, lack of supportive governmental regulations, and loss of revenues to hospital gift shops.

84–1883. Botvin, G. J.; Baker, E.; Renick, N. L.; Filazzola, A. D.; Botvin, E. M. **A Cognitive-Behavioral Approach to Substance Abuse Prevention.** *Addictive Behaviors* 9(2): 137-147, 1984.

The effectiveness of a 20-session cognitive-behavioral approach to substance abuse prevention was tested on 1,311 seventh grade students from 10 suburban New York junior high schools. Subjects were predominately white, from middle-class families. Pretest and posttest data were available for 1,185 students. The prevention strategy attempted to reduce intrapersonal pressure to smoke, drink excessively, or use marihuana by fostering the development of general life skills and teaching students tactics for resisting direct interpersonal pressure to use these substances. The relative effectiveness of this type of prevention program when implemented by either older peer leaders or regular classroom teachers was also compared.

Results indicate that the program had a significant impact on cigarette smoking, excessive drinking, and marihuana use when implemented by peer leaders. Significant changes were also evident with respect to selected cognitive, attitudinal, and personality predisposing variables in a direction consistent with nonsubstance use, providing support for the efficacy of a broad-spectrum smoking prevention strategy and tentative support for its applicability to the prevention of other forms of substance abuse. (Auth. Abs. Mod.)

84–1884. Botvin, G. J.; Renick, N. L.; Baker, E. **The Effects of Scheduling Format and Booster Sessions on a Broad-Spectrum Psychosocial Approach to Smoking Prevention.** *Journal of Behavioral Medicine* 6(4): 359-379, December 1983.

The effectiveness of a 15-session psychosocial smoking prevention strategy was tested on 902 seventh graders from seven junior high schools in suburban New York over 2 years. The five components of the prevention program, implemented by regular classroom teachers, were (1) cognitive, dealing with the immediate consequences of cigarette smoking; (2) decision-making; (3) relaxation training; (4) social skills training; and (5) self-improvement. In addition to testing overall effectiveness, the relative efficacy of two different scheduling formats was compared and the extent to which "booster" sessions conducted during the year after completion of the program helped to maintain reductions in new smoking was also examined. Results indicated that the prevention program reduced new cigarette smoking by 50 percent at the end of the first year and by 55 percent at the end of the second year for the intensive format condition. New regular cigarette smoking was reduced by 87 percent in the second year for students in the booster condition. Significant changes consistent with nonsmoking were also evident on several cognitive, attitudinal, and personality variables. (Auth. Abs. Mod.)

84–1885. Catford, J. C.; Dillow, I.A.P.; Nutbeam, D.; Waters, W. E.; Woolaway, M. C. **Public Health Education Campaigns Are Worth While. (Letter).** *British Medical Journal* 288(6417): 639-640, February 25, 1984.

In reply to a recent report (which did not refer to public health education programs against smoking) that suggested mass media initiatives in general are not worthwhile, several campaigns are reviewed, noting that results often depend on amount spent. Antismoking campaigns in Australia, Finland, and the United States are cited as showing significant sustained declines in smoking prevalence in participants compared with controls. Individual and group education is certainly needed (influence of health professionals is largely underused) but is also the most expensive on a population basis. A mass media campaign's success depends on selection criteria, just as treating a chest infection with an antibiotic depends on selection of the appropriate composition and its frequency and intensity of administration, combined with other treatment, to be successful.

84–1886. Doellner, T. **Principles for a Nationwide Long Term Smoking Prevention Program.** In: Hobbs, P. (Editor). *Public Education About Cancer. Recent Research and Current Programmes.* UICC Technical Report Series, Volume 77. Geneva, 1984, pp. 67-77.

Data from the antismoking campaign "Farvel og Tobak" (Goodbye Smoking) sponsored by the Danish Cancer Society show that in 1980 fewer children aged 9 to 15 had tried smoking and fewer were regular smokers than in 1972; however, consumption was higher among those who did smoke. The program is based on 12 principles of applied educational/psychological research and techniques derived from social marketing and communication theories. It is recognized that human consciousness works with pluses, and that primary prevention is not to prevent and combat something negative (smoking), but to make people do something that is healthier than the thing (smoking) that one is trying to prevent. Working with the primary target group (children) and the derived target group (parents, teachers, coaches), the campaign's objective was to communicate information and techniques so that people would work toward changing lifestyle, ideas, and habits. People in the derived target groups influence children through pro-health elements that enhance and stress correct behavior rather than utilizing typical antismoking campaign elements that condemn children for wrong behavior. The program is especially important in Denmark where there are no official antismoking agencies, no restrictions on tobacco sales to minors, and no health warnings on cigarettes or advertisements (and only voluntary agreement on advertising content).

84–1887. Gillies, P. A.; Wilcox, B. **Reducing the Risk of Smoking Amongst the Young.** *Public Health* 98(1): 49-54, January 1984.

Results indicate that among 136 children (aged 11 to 14) from three primary schools participating in the health education project, there was a significantly lower incidence of experimentation with smoking up to 2 years after participation. Students (74 boys and 62 girls) were matched with 134 controls from schools that did not participate. Information on smoking behavior was obtained by questionnaire before the project began and at yearly intervals. No statistically significant differences were recorded between experimental and control groups in smoking behavior at preproject testing; only 5 percent of boys and 1 percent of girls in the experimental group smoked. In the control group, the proportions were 7 percent and 2 percent, respectively. Both boys and girls in the control group were found to have over twice the risk of acquiring smoking experience compared with those in the experimental group, a trend that could not be attributed to differing parental example. Experimental group children also had increased knowledge of smoking and some of its hazards 1 year after participation, indicating that knowledge was retained over a reasonable length of time. Findings provide encouragement for those committed to the prevention of smoking in a school setting, particularly since the antismoking component comprised only one part of the project.

84–1888. Green, L. W. **Health Promotion Research and Development.** *Alabama Journal of Medical Sciences* 21(2): 217-219, April 1984.

The U.S. Public Health Service has established national priorities for health promotion (reduction of smoking is at the top of the list), but such objectives should also be a basis for private sector action, since deregulation and cuts in Federal funding may make the private sector one of the prime agents of health promotion. For any community or company health promotion effort, alcohol misuse and smoking should be the primary targets. Alcohol-related economic costs may be more than 100 billion dollars/year, and insurance and other indirect costs are higher for companies that employ smokers. These costs may increase as more women enter the work force, because more women are starting to smoke and fewer are quitting compared with men. Companies that have already instituted alcohol treatment programs have found that they pay for themselves; major commercial insurance carriers estimate that 5 dollars are ultimately saved for every dollar spent for such programs. Educational intervention for hypertension (more pervasive than alcoholism) can also be cost effective, while evidence on the cost effectiveness of physical fitness programs is tenuous. The shorter workweek, increase in number of retirees, changing structure of the family, and longer lifespans are problems that must be dealt with in assessing the community needs for health promotion and research. Although it is encouraging that cigarette and alcohol taxes exist, the Government becomes increasingly dependent on the revenues and protective of the industries, reluctant to restrain advertising, distribution, or subsidies. Thus the rebirth of voluntary community organizations interested in the area of health promotion is an encouraging and optimistic sign.

84–1889. Hag, F.-L. (Editor). **The SPAXIAN Quarterly Journal of the Society for the Prevention and Control of Smoking (SPACS).** Dhaka, Bangladesh, Society for the Prevention and Control of Smoking, January 1984, 24 pp.

The concept of SPAXISM and the policies, programs, and contentions of the SPACS organization are presented. The aims of the society are to (1) make the public aware of the health hazards of cigarettes, and (2) build a fraternity of nonsmokers who will campaign politically and in other ways against the use of tobacco. The SPACS program includes encouraging social resistance to smoking, organizing nonsmoking groups among youth, and coordinating with other agencies to promote health and control the smoking habit. A summary of the health hazards of smoking, an overview of a proposed antismoking campaign in Bangladesh, and a list of the society's board of directors for 1984 are provided.

84–1890. Hymowitz, N.; Laquatra, I.; Karl, S. **The Smoking Cessation Reduction Action Program.** *Journal of the Medical Society of New Jersey* 81(6): 487-490, June 1984.

The Smoking Cessation Reduction Action Program (SCRAP), designed to incorporate facets of long-term community disease prevention trials into a cost-effective worksite smoking cessation program, is described. Key features of SCRAP include (1) commitment to a long-term health improvement program; (2) initial assessment of cardiovascular and lung function, carbon monoxide exposure, and medical history to help smokers personalize smoking risks; (3) evaluation of positive health changes throughout followup; (4) teaching cessation and abstinence skills; and (5) health education activities. An initial pilot project will be carried out in seven hospitals in three New Jersey counties. Since only three randomly selected hospitals will implement the health education and organizational management components, it will be possible to assess their effects on initial and long-term cessation rates. Information gathered during the pilot phase will be used to develop manuals, under the supervision of the American Heart Association, that will cover the training of smoking counselors, group intervention procedures, health education, and organizational management. After initial employee participants have been followed up for 1 year, SCRAP will be evaluated and may then become available for implementation in other worksites.

84–1891. Panton, R. S. **Can the Pharmacist Help the Smoker?** *Pharmaceutical Journal* 282(6381): 698-700, June 9, 1984.

A 6-week antismoking campaign in three areas of the West Midlands (inner-city, suburban, rural) found that a high proportion of pharmacists who participated (73 percent) felt that trying to persuade people to stop smoking is part of their job. Pharmacists (n=77) who responded to questionnaires were offered three levels of involvement: (1) displaying posters and leaflets; (2) providing a slide program for young children in addition to displays; or (3) adding a machine to measure carbon monoxide in expired air. Most participants chose level 1 activity, and 50,000 booklets were given out, generally accompanied by discussion or advice. The majority felt the campaign was effective, but it was suggested that future campaigns should provide materials designed for use in limited space, more local publicity, and more time (or be continuous) so that the pharmacy would always be seen as an antismoking area. The slide program was well received, and the monitoring machine created a great deal of interest (approximately 5,000 people were screened). Many pharmacists noted that antismoking advice is most effective when given to people with smoking-related symptoms.

84–1892. Polich, J. M.; Ellickson, P. L.; Reuter, P.; Kahan, J. P. **Strategies for Controlling Adolescent Drug Use.** Santa Monica, California, Rand Corporation, No. R-3076-CHF, February 1984, 221 pp.

Findings of an 18-month review of the most promising approaches to controlling drug use among adolescents are reported, based on recent research in smoking prevention. Data on the nature of substance use are presented, and the possible effectiveness of law enforcement, treatment, and prevention programs in dealing with the problem is assessed. Availability, patterns of cigarette and other drug use, and the principal drugs consumed are examined, as are current cessation programs and future prospects for treatment. Drug law enforcement is reported with respect to enforcement against retail dealers, attacks at the distribution level, reduction of foreign crop production, and need for formulating guidelines and policies. Primary prevention through education, considered the most promising route, is discussed in terms of social- vs. information-oriented education, targeting, current smoking prevention programs as models for other drug education, and the efficacy of prevention programs.

84–1893. Public Health Reports. **Seven Prevention Projects Aimed at Behavior Changes by Children and Teenagers.** *Public Health Reports* 99(1): 39-40, January-February 1984.

"Project Model Health," a 2-year effort by the Wisconsin Department of Health and Social Services, will attempt to influence high school students with regard to cigarette smoking, marihuana use, drinking and driving, diet choices, and sexual activity. College students will be used as classroom leaders. Students in the special curriculum will be compared with a control group. The other projects, which do not directly specify smoking as a behavior, will focus on the development of strategies to encourage healthy lifestyles among children and youth. Programs, funded by the Public Health Service, will be carried out by the John F. Kennedy Institute for Handicapped Children (Baltimore); the American Academy of Pediatrics; the University of Colorado Health Sciences Center; the Department of Health of American Samoa; the Colorado State Health Department; and the University of Houston and the Galveston County Health District.

84–1894. Whelan, E. M. **A Smoking Gun: How the Tobacco Industry Gets Away With Murder.** Philadelphia, George F. Stickley Company, 1984, 263 pp.

A three-part examination of the tobacco industry is presented, exploring how the industry developed, thrived, and created a chain of economic and physical dependence. Part one describes the health risks of tobacco, the paradox of the smoking habit in the health-conscious 1980s, and the current ploys used by the tobacco industry to negate the scientific evidence. Part two traces the launching of the cigarette, its initial rejection by those accustomed to the pipe and cigar, and its ultimate success. The corresponding gradual accumulation of data on tobacco's health effects and the tobacco industry reaction are also documented. The third section presents mandates for action against the smoking-related disease epidemic that include (1) forcing smokers to carry the economic burden of the habit, (2) pursuing tobacco industry litigation, (3) ending the exploitation of developing countries, (4) banning cigarette advertising, (5) protecting nonsmokers, and (6) increasing public awareness. An overview on smoking cessation programs is provided, and three appendices (tobacco's industrial network, helpful organizations, and recommended reading) are included.

See also, **84–1708, 84–1801, 84–1879**

SMOKING CESSATION METHODS

84–1895. Blum, A. **Nicotine Chewing Gum and the Medicalization of Smoking.** *Annals of Internal Medicine* 101(1): 121-123, July 1984.

It is claimed that there is insufficient evidence that nicotine chewing gum is effective in smoking cessation, and the Food and Drug Administration (FDA) is criticized for approving it. The FDA rejected 12 of the 14 studies submitted by Merrell Dow Pharmaceuticals, Inc., for either lack of efficacy or critical flaws. The two remaining studies showed evidence of efficacy, but did not meet the standards for FDA approval. However, after submission of two new studies, and despite questions about the safety and efficacy of the gum, the FDA ruled that short-term exposure to nicotine via prescription gum is preferable to cigarette smoke exposure and that the gum increased the likelihood of quitting among participants in "acceptable counseling programs." The FDA is also criticized for presuming that smoking is synonymous with addiction to nicotine, reinforcing the notion that smoking is a medical problem with prescribable solutions. The manufacturers do not suggest that the gum will work in a person who is not highly motivated. Nicotine gum is not a panacea, and the effects of the physician's caring attitudes, financial incentives, and antismoking reinforcement in cessation warrant continued support.

84–1896. Bredehoft, W. P. **Smoking Abstention or Relapse: The Role of Causal Attributions and Self-Efficacy Expectations.** Doctoral Dissertation, Boston University, University Microfilms International 83-09749, 1983, 99 pp.

Results indicate that while attribution and self-efficacy expectation variables alone are not powerful enough to accurately predict relapse, when combined with other variables they add to the understanding of the cessation process and increase predictive power. To determine the predictive power of attribution and self-efficacy in recidivism after smoking cessation, 29 men and 54 women were surveyed 2 days after initial abstinence in 1 of 16 quit-smoking programs and at 3-month followup. No differences were observed in relapse rate or in subject characteristics between the different cessation programs. Analysis of questionnaires revealed that relapsers were more likely to attribute initial success to external causes (luck and task difficulty) than were abstainers. Relapsers did not differ from abstainers on the self-efficacy measures, although adding self-efficacy variables to the significant demographic variables caused a significant increase in predictive power. An a posteriori finding was that relapsers appeared to be overly confident of their initial success, rating the need to smoke lower than did continuing abstainers. Relapsers were found to be less educated, older, and less likely to be married. No differences between the groups were found on the hypothesized smoking variables, but relapsers unexpectedly reported fewer previous attempts to quit smoking than abstainers. (Auth. Abs. Mod.)

84–1897. Brod, M. **Stress, Coping, and Smoking Cessation.** Doctoral Dissertation, University of California, San Francisco, University Microfilms International 84-00023, 1983, 112 pp.

The hypothesis that initial nicotine intake, self-efficacy expectations, and social support would mediate the effects of stress and coping during smoking cessation was not supported by a study of 111 smokers participating in a smoking treatment program. Smokers (55 females, 56 males; average age 38) were randomly assigned to either behavioral, nicotine chewing gum, or combined treatment programs. Subjects were predominantly Caucasian (86 percent), middle to upper-middle class (88 percent), and single (52 percent), and had more than a high school education (68 percent). Measures of stress and of coping were taken at pretreatment, end of treatment, and at 6-month followup. Stress was assessed with the Schedule of Recent Experience and the Daily Hassles Scale. Coping was assessed with the Ways of Coping Scale and by the Smoker's Coping Scale, which were developed for this study. Quitters during treatment were found to use more coping strategies that they considered successful. Continuing smokers used more self-defined unsuccessful strategies. Considerable overlap in both cognitive and behavioral strategies used by the two groups were found. Frequency of coping efforts was the same in successful and unsuccessful quitters. A trend was found suggesting a synergistic effect of major life stress and minor daily hassles on smoking status. However, there was no evidence to support the hypothesis that life changes or daily hassles during treatment result in lower quit rates. Possible explanations for the negative findings are offered. (Auth. Abs. Mod.)

84–1898. Brodoff, A. S. **Smoking Cessation. What Role for the New Nicotine Gum?** *Patient Care* 18(11): 196-198, 200, 203, June 15, 1984.

Nicotine chewing gum provides nicotine blood levels comparable to those in smokers, thus controlling withdrawal symptoms. Candidates for prescription of the gum include patients with obstructive lung disease and those at high risk of smoking-related illness. Patients with cardiovascular disease or temporomandibular joint disease and women who are or will be pregnant or nursing should not be given the gum. The Fagerstrom inventory can determine a smoker's need for nicotine gum (the higher the score, the more likely the gum will be of benefit). However, the gum is best used as an adjunct to a formal behavior modification smoking cessation program. It is only an aid, and will not work unless the smoker is committed to quitting. Counseling and supervision by the physician remain important. Cigarette consumption should be reduced to 12 to 15 cigarettes/day prior to setting a target date for beginning gum use. The gum should be chewed 15 to 20 minutes before an anticipated craving for a cigarette. Use of the gum usually subsides naturally over a period of 2 months. Side effects may include mouth irritation, stomach upset, light-headedness, hiccups, or jaw muscle ache. Quit rates near 50 percent have been achieved with use of the gum in cessation programs.

84–1899. Christen, A. G.; McDonald, J. L., Jr.; Olson, B. L.; Drook, C. A.; Stookey, G. K. **Efficacy of Nicotine Chewing Gum in Facilitating Smoking Cessation.** *Journal of the American Dental Association* 108(4): 594-597, April 1984.

After 6 weeks of using nicotine (3 mg) chewing gum, 36 of 105 smokers (34.3 percent) were abstinent compared with only 11 of 103 subjects (10.7 percent) given placebo gum. Subjects, dental patients who expressed a desire to stop smoking, were followed up for 15 weeks. Both the placebo and experimental groups showed a 25 percent dropout rate, with those leaving the study citing reasons unrelated to the nicotine gum. At 15 weeks, average gum consumption had declined to three pieces/day, reflecting lack of motivation and absence of a maintenance component in the study design. Correspondingly, the number abstinent at 15 weeks had also declined (13 of the nicotine group vs. 5 of the placebo subjects). Throughout the study, however, the nicotine group had significantly higher quit rates than the placebo group. Gum was well tolerated, with reported adverse effects (ulcers, jaw muscle ache, nausea, hiccups, and gastrointestinal symptoms) similar but higher in the nicotine gum group than in the placebo group.

84–1900. Christenson, P. D. **Quantitative and Qualitative Results of a Utah Smoking Risk Reduction Program.** Doctoral Dissertation, University of Utah, Salt Lake City, University Microfilms International 84-00463, 1984, 184 pp.

The project replicated a Centers for Disease Control school-based health education curriculum for sixth grade students, modifying evaluation procedures to assess the program both quantitatively and qualitatively. Overall results established the effectiveness of the Smoking Education Risk Reduction program in teaching knowledge of the health consequences of smoking, decisionmaking skills, and resistance-to-persuasion skills. Measures of health knowledge, decisionmaking skills, resistance-to-persuasion skills, tobacco use, and self-report categorization for cigarette smoking were analyzed quantitatively; program implementation, participant views of the program, and effects were assessed qualitatively; and quantitative data were analyzed in terms of consistency with qualitative data. Participating teachers were randomly assigned to either experimental or control groups. Significant group differences were found on the Smoking Information Test between experimental and control subjects. Differences between groups were also significant for decisionmaking skills. Although gender differences were found on selected categories of the Health Decision-Making Index, such differences were not seen in specific resistance-to-persuasion skills, which did, however, show significant group differences. No significant group differences were found between the experimental and control teachers in knowledge of the health consequences of smoking. Qualitative results indicated that most teachers, principals, and students viewed the program positively, but demonstrated the need for reexamination of all instrumentation used to evaluate the program, and the need for a close working relationship between teachers and local health educators. (Auth. Abs. Mod.)

84–1901. Clavel, F.; Benhamou, S. **Desintoxication Tabagique. Comparaison de l'Efficacite de Differentes Methodes. Resultats Intermediaires d'une Etude Comparative. [Detoxication of Tobacco-Addicts by Different Methods. Intermediate Results of a Comparative Study.]** *Presse Medicale* 13(16): 975-977, April 14, 1984, French.

A comparative study to evaluate the effectiveness of different methods of smoking cessation is currently in progress. In this study, the percentage of subjects who gave up smoking after 1 month and changes in this percentage over a 12-month period are being determined in a group treated with acupuncture, a group treated with nicotine chewing gum, and a control group. Results of an intermediate analysis include 45 percent of the total number of subjects required. No statistically significant difference between the control group and the two treatment groups is evident. Volunteers are still being recruited. (Auth. Abs.)

84–1902. Davis, J. R. **Relapse Prevention and Smoking Cessation.** Doctoral Dissertation, Wayne State University, Detroit, University Microfilms International 83-15586, 1984, 562 pp.

Specific high-risk situations and effective coping strategies of successful ex-smokers were identified, a role-play assessment instrument was constructed, and its reliability investigated with 45 subjects assigned to either control, enhanced control, or experimental groups. Controls received only a broad-spectrum group treatment; enhanced controls discussed situations that posed a high risk for relapse in addition to receiving the group treatment; and experimental subjects received the group treatment, social skills training to enhance coping skills in high-risk situations, and problem-solving training to promote generalization. Pretreatment, posttreatment, and 3-month followup measures included self-monitored and self-reported smoking rates, exhaled air carbon monoxide, a role-play measure of coping skills, and paper-and-pencil measures of expectancies and smoking-related information. Groups did not differ on any variable in pretreatment analysis. Posttreatment analyses showed that subjects in all groups reduced smoking rates significantly and that groups did not differ in number of quitters. The experimental group subjects increased coping skills significantly, tended to take longer to relapse, and smoked fewer cigarettes at time of relapse. Thus, a modest effect for the experimental treatment was shown; however, for subjects in all groups, once a relapse occurred, regular smoking was resumed. (Auth. Abs. Mod.)

84–1903. Frydrychowski, A.; Ostrowska, B. **Krotkoterminowe Wyniki Leczenia Nikotynizmu Metoda Akupunktury. [Acupuncture in the Treatment of the Smoking Habit.]** *Psychiatria Polska* 18(1): 31-34, 1984, Polish.

In an evaluation of the effectiveness of acupuncture for cessation of smoking treatment, experiments were carried out on 60 patients. Punctures were made at six acupuncture points of the body and three points in the ear concha. Positive results were obtained in 70 percent of the patients, and 40 percent gave up smoking completely. (Auth. Abs.)

84–1904. Hughes, J. R.; Hatsukami, D. K.; Pickens, R. W.; Krahn, D.; Malin, S.; Luknic, A. **Effect of Nicotine on the Tobacco Withdrawal Syndrome.** *Psychopharmacology* 83(1): 82-87, April 1984.

Treatment of tobacco withdrawal with nicotine gum suggests that the tobacco withdrawal syndrome is caused, in part, by nicotine deprivation. Signs and symptoms of tobacco withdrawal were measured in 99 smokers (54 males and 45 females, average age 36.7 years) who fulfilled DSM-III criteria for tobacco dependence and a past history of tobacco withdrawal. After baseline measurement, subjects were randomly assigned to receive either nicotine or placebo gum in a double-blind manner. Subjects stopped smoking, chewed gum freely, and returned for measurements on the first, second, and fourth evenings of abstinence. Nicotine reduced the increase in irritability, anxiety, difficulty concentrating, restlessness, impatience, and somatic complaints that subjects reported after cessation. Reductions were confirmed by ratings of significant others and by subjects' scores on the Profile of Mood States. Nicotine did not reduce increases in cigarette craving, hunger, eating, insomnia, tremulousness, or supine heart rate after cessation. The effects of nicotine occurred immediately and persisted throughout the study. Although many subjects correctly identified their drug group, efficacy of the gum was independent of drug identifications. (Auth. Abs. Mod.)

84–1905. Jarvik, M. E.; Schneider, N. G. **Degree of Addiction and Effectiveness of Nicotine Gum Therapy for Smoking.** *American Journal of Psychiatry* 141(6): 790-791, June 1984.

Nicotine chewing gum was more helpful in smoking cessation to subjects who registered as highly dependent on nicotine (7 or higher on the Fagerstrom tolerance questionnaire). In a double-blind trial, 48 heavy smokers (randomly assigned to either 2 mg nicotine gum group or placebo gum group) were followed up daily the first week of cessation, weekly for 4 weeks, and at 3, 6, and 12 months after cessation. At each abstinence check, subjects were given half an hour of individual clinical support, then completed questionnaires. Pulse, body weight, and carbon monoxide breath level were also measured. Among the highly dependent smokers, only 8 percent of those receiving placebo compared with 41 percent of those receiving active gum remained abstinent. Of those with low dependence scores, 30 percent of the placebo vs. none of the active gum group remained abstinent. Although in the expected direction, differences were not statistically significant.

84–1906. Medical Letter on Drugs and Therapeutics. **Nicotine Gum.** *Medical Letter on Drugs and Therapeutics* 26(601): 47-48, May 11, 1984.

Nicorette, a Merrell Dow product recently approved by the U.S. Food and Drug Administration (prescription only), contains 2 mg nicotine bound to an ion-exchange resin in a gum base. Nicotine is absorbed through the buccal mucosa as the gum is chewed; blood nicotine concentrations after 1 hour are about half those attained by smoking. Nicotine is metabolized mainly by the liver and metabolites are excreted by the kidneys; unreleased nicotine is excreted in stool. The gum reportedly relieves smoking withdrawal symptoms (e.g., irritability and concentration difficulty). In controlled trials, nicotine gum chewers have had 1-year abstinence rates from 1.5 to 2.2 times higher than controls. Reported side effects include burning or soreness of the mouth and throat and gastric distress. Dosage varies according to user; the manufacturer suggests 10 to 12 pieces/day for about 3 months. Nicorette costs the pharmacist 14.21 dollars/pack of 96 pieces. It is concluded that nicotine gum may help some people quit smoking.

84–1907. Ossip-Klein, D. J.; Shapiro, R. M.; Stiggins, J. **Freedom Line: Increasing Utilization of a Telephone Support Service for Ex-Smokers.** *Addictive Behaviors* 9(2): 227-230, 1984.

In an investigation of various noncommercial promotional strategies to attract callers to a telephone support system (the Freedom Line) aimed at enhancing maintenance of nonsmoking, it was found that high utilization of a taped telephone support service for ex-smokers can be achieved at minimal expense. A practical concern was whether utilization would be great enough to warrant long-term clinical implementation. A two-pronged approach was used to promote the service: (1) through agencies with existing channels for community ties with smokers and ex-smokers (e.g., American Lung Association) and public service announcements on multiple radio stations; and (2) intermittent, but ongoing, updated newspaper coverage and television/radio interviews. When the two approaches were alternated monthly, approximately three times as many calls were received during the second type of promotion ($p<0.001$). Over an 8-month period, more than 8,000 calls were received. Achieving and maintaining high utilization opens the door for evaluating the Freedom Line's effectiveness.

84–1908. Pomrehn, P. R. **Nicotine Gum as an Adjunct in Smoking Cessation.** *Iowa Medicine* 74(6): 259-260, 262, June 1984.

Nicotine gum, now available by prescription (2 mg/piece only), is an effective adjunct for smokers attending cessation clinics, but results of use outside this context are less clear. However, physicians play an important role in helping patients stop smoking, and nicotine gum seems to be an additional means, along with advice, self-help materials, and group programs, to improve chances of success. To help physicians and pharmacists respond to patients' questions about the gum, the pharmacology and pharmacokinetics of nicotine vs. nicotine gum are described. Several clinical studies indicating that nicotine gum improved quit rates are reviewed. Possible side effects are given, and instructions on how to use the gum and how often it can be chewed are provided. Patients who use the gum for 2 to 3 months or more appear to be less likely to relapse, and some investigators advocate keeping the gum available for at least a year in case the urge to smoke arises.

84–1909. Rabkin, S. W.; Boyko, E.; Shane, F.; Kaufert, J. **A Randomized Trial Comparing Smoking Cessation Programs Utilizing Behaviour Modification, Health Education or Hypnosis.** *Addictive Behaviors* 9(2): 157-173, 1984.

A randomized trial to compare the efficacy of hypnosis, health education, and behavior modification programs for cessation of smoking found no significant differences with respect to the proportion who reported quitting smoking, the number of cigarettes smoked, or change in serum thiocyanate levels among 168 volunteers. Followup data 3 weeks after completion were available for 140 subjects, and each program showed significant reductions in reported cigarette consumption and serum thiocyanate levels (an indicator of long-term cigarette consumption) compared with entry and with the control group. Reported cigarette consumption ascertained 6 months later again showed no significant differences between programs. Factors such as subject age, age at starting cigarette smoking, educational level, marital status, and spouse or partner's smoking did not identify subgroups with differences between treatment responses. In the hypnosis program, 29 percent reported abstinence at 3 weeks; for health education, 32 percent reported quitting; and for behavior modification, 37 percent had quit. It was therefore concluded that all programs are equally effective for changing cigarette smoking. (Auth. Abs. Mod.)

84–1910. Schneider, N. G.; Jarvik, M. E.; Forsythe, A. B. **Nicotine vs. Placebo Gum in the Alleviation of Withdrawal During Smoking Cessation.** *Addictive Behaviors* 9(2): 149-156, 1984.

Subjects (n=24) receiving placebo gum experienced significantly more severe withdrawal symptoms than those given nicotine chewing gum (n=26) in a double-blind, placebo-controlled trial. Withdrawal symptoms were systematically scaled for 1 week by obtaining baseline responses on withdrawal items and comparing them with 5 days of smoking abstinence. There were 12 females and 14 males in the group receiving nicotine gum, and 14 females and 10 males in the placebo group. Student t tests showed no differences between groups in age, amount smoked, years smoked, prior attempts to quit, or prior incidence of withdrawal. A withdrawal scale was derived from smokers' complaints in previous abstinence attempts. Carbon monoxide determinations served to verify abstinence. Pulse rate dropped significantly during abstinence for the placebo vs. the nicotine group. All subjects experienced significant increases in withdrawal over time (no sex differences were found). It is concluded that the appearance of withdrawal symptoms may be attributed, in part, to the removal of nicotine per se, and conversely, nicotine-specific symptoms may be alleviated with nicotine gum. (Auth. Abs. Mod.)

TOBACCO PRODUCT ADDITIVES

84–1911. Boden, R. M. **Use in Augmenting or Enhancing the Aroma or Taste of Smoking Tobacco Compositions and Smoking Tobacco Articles of Branched Chain Olefinic Alcohols.** United States Patent No. 4,457,318, July 3, 1984, 33 pp.

The branched chain olefinic secondary alcohols described impart woody, oriental, and minty flavor and aroma characteristics to smoking tobaccos. Recommended proportions of flavoring additive are 250 ppm to 1,500 ppm (0.025 to 0.15 percent by weight), but up to 10,000 ppm (1.0 percent by weight) is acceptable. The compounds described are prepared by reacting branched ketones with a reducing agent in the presence of an appropriate catalyst (for example, sodium borohydride with a protic solvent such as isopropyl alcohol) and then distilling the products to the desired saturated alcohols.

84–1912. Schreck, R. P.; Hall, J. B. **Uses of Methyl Phenyl Pentanol Derivatives in Augmenting or Enhancing the Aroma or Taste of Smoking Tobacco and Smoking Tobacco Articles.** United States Patent No. 4,458,699, July 10, 1984, 27 pp.

The methyl phenyl pentanol derivatives described impart green, grapefruit-like, nootkatone-like, animalic, leathery, vetiver-like, olibanum, musky, and floral nuances to tobacco smoke aroma and flavor. Recommended amounts of the compounds are 300 to 5,000 ppm, but a concentration of 500 to 1,000 ppm by dry weight is preferable. The compounds may also be incorporated into the paper, filter, packaging, or seam paste of cigarettes.

TOBACCO MANUFACTURING AND PROCESSING

84–1913. Labbe, F.A.M.; Nowers, J. R. **Manufacture of Cigarettes.** United States Patent No. 4,459,998, July 17, 1984, 4 pp.

With the described method, cigarettes are constructed by forming a basic filler stream of tobacco, tobacco substitute, or a combination of both and folding a web of reconstituted tobacco around the filler stream. The combination is then enclosed in a continuous paper wrapper and cut into predetermined lengths. The tobacco web may be continuous or in an expanded form. Utilizing this method, which provides a simple means of adjusting various tobacco characteristics, a cigarette-making machine could produce different brands by changing to appropriate reels of reconstituted tobacco.

TOBACCO ECONOMICS

84–1914. Ahmad, M. **Exports Nil; Most Tobacco Expended in Domestic Production.** *Tobacco International* 186(14): 5-6, 8, 10, July 6, 1984.

Because of poor weather, the 1983 Pakistani tobacco crop fell nearly 4 million kg short of the 50.5 million kg required by the cigarette industry and other users, and leaf exports were halted during 1983. Cigarette exports (1,307,400 million pieces), however, were close to 1982 levels (1,374,000 million pieces). Research to improve leaf production has resulted in a 32 percent increase in yield and in better quality, and 1984 production needs are expected to be met. After recent stagnation, cigarette sales increased 3.81 percent in 1983; filtered sales rose 20 percent to a 54 percent share of the market. Also reflecting changing consumer tastes, 21 new brands were launched between January 1983 and May 1984, most in low-price categories. The cottage bidi industry, revived when the Government allowed free import of bidi wrapper leaf, is competing with low- and medium-priced brands, which has spurred the introduction of very low priced brands. Bidis are much cheaper than the lowest priced cigarettes. It is also noted that an alarming increase in drug abuse has overshadowed antismoking activities.

84–1915. Blakeney, M.; McKeough, J. **The Right to Advertise. The Cigarette Debate.** *Media Information Australia* 31: 42-46, February 1984.

Opposition to recent attempts to regulate or ban cigarette advertising in Australia is based on two freedoms fundamental to Western democracy: freedom of speech and freedom of enterprise. Commercial groups having an interest in the continuation of lucrative cigarette advertising revenues are self-appointed defenders, but their unusual championship of the freedoms of speech and enterprise compels an examination of the extent to which they extend these same freedoms to those who wish to warn about the dangers of cigarette advertising. Arguments are characterized by the same intellectual dishonesty used to advertise cigarettes; freedom of speech is countervailed by freedom from defamation, blasphemy, obscenity, and sedition, and arguments related to cigarette advertising are dishonest in that they deceive by not revealing the whole truth. The freedom of enterprise is subject to the more important freedom from abuse, and despite denials, it is incontrovertible that smoking damages health. It is a greater infringement of liberty that taxpayers support the medical and social consequences of smoking through income tax than to have to endure legislation aimed at decreasing the habit and its cost to the community. The tobacco industry is a highly organized and powerful entity whose expertise may well derive from its ability to convince large numbers of consumers that its carcinogenic, addictive, noxious, and foul-smelling products are socially acceptable, masculine, feminine, ruggedly individual, egalitarian, elitist, healthy, fresh, mild, and sexually potent.

84–1916. Gloede, W. **Cigaret Marketers Come Out Smokin' With Innovations.** *Advertising Age* pp. 1, 98, June 18, 1984.

Marketing activity in the cigarette industry is on the upswing following 18 months of relative calm after the Federal excise tax was doubled. The increase cost the industry 5 to 6 percent in total unit sales last year and has brought about changes. Recently, Philip Morris (Marlboro is the largest selling brand in the United States with 20 percent of the market) began testing a 25-cigarette pack; Brown and Williamson will enter the generic and private label category; R. J. Reynolds will market Doral as a branded item at a generic price; Liggett and Myers will test a new midprice brand; Lorillard is expanding its Newport line; and American Tobacco is taking orders for Lucky Strike Lights and Lucky Strike Lights 100s. Much of the new market activity has been spurred by Liggett and Myers' spectacular success in the generic and private label categories. It is speculated that generics may get up to 10 to 15 percent of the market, but traditional brands will continue to capture the largest share, relying on marketing strategies and programs for support.

84–1917. Hall, R. **United States Growers Need a New Focus.** *Tobacco International* 186(13): 38, 40, 42, June 8, 1984.

U.S. tobacco production is geared to a pricing formula rather than to user needs. The price formula, originally designed to keep minimum market prices above levels disastrous to growers, may now be the single most damaging factor to the long-term economic success of growers. Tobacco must be seen as an international crop, and unless U.S. tobacco production is refocused on international economic and political realities, U.S. growers will serve an ever-smaller share of the world's tobacco users. U.S. producers have the economic ability to recapture a share of the world market. However, changes in the current tobacco program will have a strong impact on growers in other countries, who have a greater economic investment in tobacco production than growers in the United States. Also, issues are complicated by political systems that seek to increase domestic production and exports while limiting domestic consumption and imports. The prime reform needed is to recognize the right to income from possession of an allotment rather than the actual production of tobacco. Cuts in U.S. poundage allotments may be regarded as a successful agricultural foreign aid program on the part of U.S. growers in recent years.

84–1918. Harman, A. **Negotiators Ask: Where Do We Go From Here?** *Tobacco Reporter* 111(6): 56, June 1984.

On May 18, Ontario tobacco growers rejected the Canadian Tobacco Manufacturers Council offer for a total crop size of 170 million pounds with a minimum guaranteed average price of 1.65 Canadian dollars. Manufacturers had originally asked that the crop size be cut to 145 million pounds (a drop of about one-third), potentially forcing 800 growers out of business, canceling 15,000 seasonal jobs, and reducing revenues by 114 million Canadian dollars. The proposed cut in crop size is a result of foreign competition, reduced domestic demand, declining exports, and 10 million pounds from last year's crop still unsold. Whether an agreement can be reached before planting time remains to be seen, but in the meantime, some 2,000 of Ontario's 2,500 tobacco growers have asked the Ontario Flue-Cured Tobacco Growers' Marketing Board to seek protection under the National Farm Products Agencies Act. Inclusion of tobacco under the act would allow growers to set prices based on production costs and give them power to limit imports, protecting them from market fluctuations and the dictates of manufacturers.

84–1919. Kille, T. **In Zimbabwe, Small-Scale Burley Plans Persist, But Bigger Better.** *Tobacco International* 186(11): 14-15, May 25, 1984.

Rhodesian Burley production started in earnest in the late 1950s and expanded rapidly owing to its suitability as a replacement for maize as a small farm cash crop and its lack of need for a high level of management. After the Unilateral Declaration of Independence in the 1960s, new growers (European producers who wanted to escape from flue-cured quotas) brought expertise to Burley growing and handling, and the crop was improved. Production declined, however, when quotas were eased and growers returned to flue-cured. The Burley Tobacco Consortium, formed in the 1970s, brought stability, and crop values have increased steadily. The industry has a core of 100 large-scale commercial producers planting an average of 11 ha to Burley. Some small-scale commercial growers are reaching 4 ha, but most plant only slightly more than the peasant (communal) farmers, who grow about 0.4 ha. Since the mid-1970s, rising quality has enabled large commercial growers to increase average prices, while those of the small-scale and peasant producers have declined. Total Burley production in 1983 exceeded 4.5 million kg, with about 0.8 million kg from peasant farmers and about 0.20 million kg from small-scale commercial growers.

84–1920. Marshall, J. P. **Tobacco Marketing Issues.** In: Pugh, C. R. (Editor). *Tobacco Issues for the 80's.* Proceedings of the Conference of the Colleges of Agriculture in the Five Flue-Cured States, March 8-9, 1983, Myrtle Beach, South Carolina. Available from C. R. Pugh, Agricultural Extension Service, North Carolina State University, Raleigh, 1983, pp. 87-94.

Tobacco marketing problems have been created by the congressional mandate for a no-net-cost program and the decrease in available quota, increases in stocks under loan, increase in price supports, and the shift from family farm to commercial production (increasing the proportion of quota leased). Potential solutions to problems in the Lease-and-Transfer Program include (1) returning quota ownership to growers; (2) increasing quota to reduce cost of leasing; (3) administratively terminating the program; (4) improving market information about available quota; and (5) establishing a Lease-and-Transfer Elimination Fund. Marketing of cured leaf will also be more closely monitored, resulting in strengthened controls on nesting, quality, supply, the quantity of leaf held by Stabilization, and the length of time leaf is held and an examination of the charges paid for selling the crop. Marketing of leaf held by producer-owned cooperatives will also be evaluated in terms of selling the stocks in Stabilization via traditional channels, or by price reduction to market-clearing levels (possibly moving producers toward acceptance of a two-price system). U.S. growers have functioned as a cartel, but the quantity and quality of foreign-produced leaf now present a challenge to improve U.S. quality and lower costs throughout the production process. Protectionist measures and price-cutting wars in the world market would be self-defeating.

84–1921. Misdorp, S. **Aggressive Advertising Helps Galaxy Make Headway in Uruguay.** *Tobacco International* 186(13): 72, June 8, 1984.

An aggressive advertising war between an international brand of cigarettes (Galaxy) and a national brand (Coronado) has helped Philip Morris achieve a 30 percent share of the Uruguayan market (up from only 8 percent in 1979). However, the Government may not tolerate the forcefulness of the campaign much longer, and there are fears that further controls on cigarette advertising may be imposed. In August 1983, manufacturers were required to add health warnings to all packages and advertisements. The world recession and contraband cigarettes have also affected the market; cigarette sales declined by 30 percent compared with the average of the last 12 years. Brazilian cigarettes can be bought for half the price of national brands. It is noted that 90 percent of all cigarettes produced in Uruguay are filter tipped; 80 percent are blond, 10 percent dark, and 10 percent are roll-your-own.

84–1922. Misdorp, S. **Rain-Reduced Argentine Virginia Crop Excellent; Cigarette Sales Rise.** *Tobacco International* 186(13): 35, 37-38, 40, 42, June 22, 1984.

The quality of Argentine tobacco is the best in 10 years, with total production for the 1983-1984 season estimated at 77,440 tons (40,200 tons flue-cured, down from 1982 levels owing to heavy rains, and 17,300 tons Burley). However, prices paid to farmers are the lowest in 30 years. Low prices, combined with a monthly inflation rate of 18 percent and slow repayments by the Tobacco Fund, have resulted in severe financial problems for farmers. Growers protested the slow repayments by blocking major highways with tractors, and objected to the low prices. After an anticipated radical change in governmental financial policies did not materialize, new prices were released. Because of increased salaries and a reduction in taxes, cigarette sales rose 6.5 percent during 1983, after declines of 8.8 percent in 1982 and 7.2 percent in 1981. Also contributing to the sales boost was the introduction of several new low-price brands during 1983. No advertising restrictions exist in Argentina, but requests have been made in Congress to prohibit cigarette sales to youth under age 18, to give preference to nonsmokers seeking public jobs, and to forbid smoking in Congress. In response, Salta and Jujuy, two provinces that depend almost entirely on tobacco, are planning intensive counter campaigns to such antismoking efforts.

84–1923. Morris, J. E. **This Tobacco Business, Part XX: Exhibitions and Exhibitors.** *Tobacco International* 186(13): 7-10, 13-14, 16-18, 20, 22-24, 26-27, 29-30, 32, 35-36, June 8, 1984.

The tobacco industry has not been a communicative industry, and fierce, ruthless competition has been an accepted way of life; however, antismoking campaigns and tax increases threatening the industry have created the need for more and better international communication. Until recently, trade journals have been the only communication link providing information (without revealing trade secrets). In the late 1960s, a successful international exhibition brought suppliers and manufacturers together, and problems in developing countries prompted the addition of a conference featuring speakers with specialized knowledge at the Richmond Exhibition in 1983. Response confirmed that the tobacco industry was seeking acceptable means of communication to resolve problems not discussed openly, and a similar exhibition and conference will be held in Richmond in 1986 (sponsored again by *Tobacco International*). *World Tobacco* sponsored an exhibition at the Hague in April 1984, and while well accepted by visitors, it attracted few representatives from developing countries. Comments by attendees are included, and although some suppliers consider multiple exhibitions an unnecessary expense, such shows do improve communication. Photographs and extensive replies from exhibitors at the Hague reflect the international flavor of the show, with quotes from representatives from Nicaragua, England, Yugoslavia, Belgium, Italy, Uruguay, West Germany, the United States, Switzerland, the Netherlands, Greece, Turkey, Canada, Brazil, Lichtenstein, India, Holland, and Austria.

84–1924. Nares, P. **Colombian Market Again Flooded With Contraband Cigarettes.** *Tobacco International* 186(13): 69, June 8, 1984.

The economic impact of smuggled American cigarettes on Colombia involves not only the tobacco industry but also the Government, which derives about 10 percent of the revenue of provincial departments from cigarette taxes. Relaxation of the Government's clampdown has meant smuggled cigarettes may have recaptured as much as 30 percent of the market. Corruption in the customs department is considered a major factor, and tobacco interests urge the current director to continue efforts to reform the service. Tobacco officials also urge the Government to protect industry jobs (particularly since unemployment is now almost 14 percent) by revitalizing the anticontraband measures.

84–1925. Nares, P. **Colombia: Anti-Smoking Drive Seems Tabled.** *Tobacco International* 186(13): 42, June 22, 1984.

The recent award of the National Quality Prize to Coltabaco, the largest cigarette manufacturer, is seen as an indication that the Betancur government does not intend to introduce punitive measures against the Colombian tobacco industry. It is suggested that the Government is hesitant to move because a decline in cigarette consumption would only accentuate the already critical (about 14 percent) unemployment problem. Little has been heard about the proposed antismoking campaign.

84–1926. Shelton, A. **Spotlight on Zimbabwe: A Promise of Improved Quality.** *Tobacco Reporter* 111(6): 32-34, June 1984.

Better weather in 1984 is expected to produce one of the best flue-cured crops in recent years, probably about 110 million pounds. In early trading, prices for lower quality showed a marked decline, while middle and better grades were selling 10 to 15 cents higher than in 1983. Europe continues to dominate the export market, but recent increases in South African rail tariffs could increase costs and have a negative effect on any price increase growers might receive and seriously affect Zimbabwe's competitive position with Brazil on European markets. It is hoped that the port of Beira will soon be able to handle all exports to eliminate the need to ship through South Africa. Burley is a young sector of the industry with many problems that are currently being addressed. Domestic cigarette consumption is declining, attributed to large tax increases and generally poor economic conditions. The dry years have highlighted tobacco's position as the main source of foreign exchange, and the Government is expected to do all in its power to promote it.

84–1927. Shelton, A. **It's the Tobacco They're After.** *Tobacco Reporter* 111(7): 3, July 1984.

All over the world, high-pressure groups are trying to toughen existing tobacco advertising regulations or completely prohibit tobacco promotion. Ultimately, advocates of advertising bans want to eliminate the use of tobacco, not the existence of tobacco advertising. However, the belief that bans on advertising will reduce tobacco use is questionable. The International Advertising Association in New York found no evidence that an advertising ban affects consumption, although the absence of promotion can significantly hold back development of new and more advanced products. In the 16 countries analyzed, market trends apparent before enactment of advertising restrictions remained unchanged in the following years. In response to the antiadvertising efforts, the Advertising Federation of Australia, where tobacco is under strong attack, has launched its own campaign to remind the public that such bans can result in lost jobs across a wide spectrum of the economy.

84–1928. Shelton, A. **Smokeless Tobacco: Moist Snuff Leads American Market.** *Tobacco Reporter* 111(7): 30-31, July 1984.

Sales of moist snuff rose 7.1 percent during 1983 and 5.4 percent during the first quarter of 1984. Loose leaf tobacco sales increased 0.1 percent in 1983, but 5.0 percent in the first quarter of 1984, indicating a possible recovery from the stagnation of recent years. In contrast, sales of firm plug, moist plug, twist and roll chewing tobacco, and dry snuff declined during 1983. The total market, however, declined only 0.3 percent, while figures for the first quarter of 1984 show a 2.5 percent sales growth over the same period in 1983. In 1983, 22 million U.S. consumers spent 900 million dollars on smokeless tobacco. A new "smokeless cigarette" named Favor, developed by Advanced Tobacco Products in San Antonio, is scheduled for production within a year. Smoker satisfaction will come from nicotine concentrate in a filter-type element. However, health advocates are now focusing on smokeless products; Ralph Nader's Health Research Group is lobbying Congress to include smokeless products in the legislative package that bans cigarette advertising. The group, citing the health hazards of smokeless tobacco products, is also petitioning the Federal Trade Commission to (1) require warning labels for smokeless products; (2) impose advertising regulations; or (3) challenge individual advertisements as deceptive or unfair.

84–1929. Spence, G. B. **Tobacco, Once a Sure-Thing Crop, Now Among the Risky in Maryland.** *Tobacco International* 186(13): 48-49, June 8, 1984.

Following 1983 tobacco crop losses, the worst in Maryland history, Farmers Home Administration offices have been besieged for 5 percent disaster loans by destitute tobacco farmers who lack money to plant new crops. Growers, who can borrow 80 percent of money lost on the 1983 crop, must put up everything they own as security, and may be required to sell everything not essential for farming operation. In addition, growers are puzzled by high demand for darker leaf (usually associated with immaturity, excessive chlorine, or pole sweat) previously considered undesirable, and are uncertain whether to harvest immature tobacco to obtain the desired color. Tobacco is no longer considered a safe income crop in Maryland.

84–1930. Sumner, D. A. **Effects of Cigarette Tax Changes.** In: Pugh, C. R. (Editor). *Tobacco Issues for the 80's.* Proceedings of the Conference of the Colleges of Agriculture in the Five Flue-Cured States, March 8-9, 1983, Myrtle Beach, South Carolina. Available from C. R. Pugh, Agricultural Extension Service, North Carolina State University, Raleigh, 1983, pp. 95-99.

Changing the excise tax causes a series of repercussions throughout the supply, demand, and market-clearing relationships in the tobacco industry. A model was developed, representing relationships between various quantities and prices, to calculate the effect of the tax increase on cigarettes. Calculations are presented that indicate the impact of the 1983 tax increase under alternative expectations of elasticities and further policy responses. Data for flexible and realistic conditions show that when plausible elasticities are used, quite small changes in the price of tobacco are implied. If exports of cigarettes or tobacco increase as prices fall, the price of tobacco declines by no more than 3 percent. The tax puts downward pressure on both price and quantity, and results depend, in part, on public policy and other market factors. With the No Net Cost program and other recent changes in the market, it will be difficult to separate the effects of the tax from other factors.

84–1931. Tobacco International. **Increased Cigarette Consumption Spurs Leaf Production in Nigeria.** *Tobacco International* 186(11): 40, 42, May 25, 1984.

Because prices for smuggled cigarettes are rising and smuggling has been reduced, consumption of domestic cigarettes has increased. The industry plans to capitalize on the surge in demand with significant expansion in 1984 and 1985, aware that many farmers want to grow tobacco commercially. Although there was a drought in 1983, the outlook for 1984 is better (early rains have already begun). However, the two manufacturers (Nigerian Tobacco Co. (B.A.T.) and Philip Morris Nigeria) are aware of long-range factors: the increasing age of the farmer population (few young farmers are growing tobacco) and irrigation systems that threaten to change growing conditions. The industry's main advantage is that it provides free seeds, chemicals, cheap fertilizer, extension services, credit, and occasionally even equipment. The country's foreign exchange situation is creating short supplies for manufacturing and reducing tobacco imports (used in blending domestic cigarettes). With all production in the hands of two companies, the Government plays a minor role and no changes are expected in the near future. There is some apprehension among industry officials that Nigeria may be chosen as a test case for a proposed World Health Organization antismoking campaign aimed at developing countries, although this is not yet definite.

84–1932. Tobacco International. **Zimbabwean Scene: Record Yield of Flue-Cured; Curing Experiments.** *Tobacco International* 186(11): 5, 7-8, 10, 12, May 25. 1984.

In spite of patchy rainfall and a high incidence of hail, record yields have been achieved and some excellent middle- and upper-stalk position leaf has been produced. The total flue-cured crop is expected to reach 110 million kg, with five of the six majority grades opengrained ripe compared with only one grade in 1983. Burley has also fared better than in 1983. Efforts are being made to overcome the effects of dry weather during curing by adding moisture to barns. Peasant farmers are also receiving assistance in land preparation, fertilization, and fumigation to increase production. Some interest is being shown in dark fire-cured tobacco and research is being conducted. The export of tobacco accounts for 20 to 25 percent of the country's foreign currency earnings and the decline in value of the Zimbabwe dollar against major foreign currencies makes Zimbabwe tobacco much more attractive on export markets. Current indications for 1984-85 are that there will be no reduction in dry land acreage, and possibly a slight increase.

84–1933. Tobacco International. **Nearly All Malawi's Leaf Exported: Prime Foreign Currency Earner.** *Tobacco International* 186(11): 26, 28, 30, 32, 34, May 25, 1984.

Tobacco is Malawi's prime foreign currency earner, and the fall in value of production in U.S. dollar terms from the 1981-1982 season to the 1982-1983 season could have a significant impact. However, with better growth projected for 1983-1984 crops, good sales are expected. Most of the tobacco produced, which includes Burley, dark fire-cured, sun air-cured, and flue-cured, is exported. Market prices affect trends in volume produced, as was seen with Burley production, which increased by 50 percent following the 1980-1981 season when it received a higher price than flue-cured. A quota system has now been introduced to control Burley production, and quality is expected to improve. Total tobacco production will be down by about 10 million kg from last season, but prices for good quality leaf are up slightly compared with last season. Production figures and average prices for different varieties are tabulated for the 1979-1980 through the 1982-1983 seasons.

84–1934. Tobacco International. **Dominican Burley Growth Increasing; Cigar Sales to U.S. Booming.** *Tobacco International* 186(13): 45-46, June 22, 1984.

Dominican Republic dark tobacco, which is neutral and easily blended with more flavorful darks, has a market in dark cigarettes. However, dark tobacco consumption is declining worldwide, and even with the recent dramatic drop in production, 40,000 bales of last year's stock are still unsold. In addition, exporters are competing against subsidized dark leaf from Europe, in an already oversupplied market. Tabacalera, the Spanish monopoly, buys 70 percent of Dominican leaf and controls the market. Spain will pay no more in 1984 than in 1983 and has set strong quality specifications. Optimistically, the Government's recent currency exchange benefit for exporters will allow higher prices for tobacco farmers without increasing export prices. Attempts to convert to Burley are being made; Tropicales is already growing both Burley and Virginia (which cannot be imported legally). The Tobacco Institute is also trying to increase cigar wrapper production. Domestic cigarette sales grew about 1 percent, with blended cigarette sales increasing rapidly at the expense of dark cigarettes. Cigar production is also rebounding, increasing 10 percent in 1983 and 20 percent in the first quarter of 1984, with about 43 million of the 46 million units produced in 1983 going to the United States.

84–1935. Tobacco Reporter. **Malaysian Tobacco: Escalating Prices Hit Cigarette Market.** *Tobacco Reporter* 111(7): 42-44, July 1984.

Cigarette consumption in Malaysia declined an estimated 10 to 15 percent in 1983 (the first decrease ever noted) owing to a 100 percent jump in taxes, other price increases, and governmental steps to discourage smoking. Because of strong opposition (advertising restrictions and tax increases), cigarette manufacturers are delaying expansion plans and are diversifying. However, most tobacco farmers are poor and belong to the Malay ethnic group that controls the government. Thus, tobacco is a politically sensitive crop and will receive continued support via high import tariffs and minimum price supports. The Ministry of Primary Industries views tobacco as a potential export item, and in cooperation with the National Tobacco Board and the National Farmers' Organization, an additional 2,000 ha will be planted to tobacco. Landless farmers and fishermen will be given leased land and financing and taught to grow and cure leaf. American blends are being produced locally, reducing imports, and the Government wants locally grown tobacco to supply 70 percent of cigarette content by 1986. Output has been increased to over 1,600 kg/ha. Details of advertising restrictions, price increases by brand name, leaf quality issues, weather conditions, and the supply and distribution of leaf stocks are provided.

84–1936. Tobacco Reporter. **Cigarette Sales: Rising Taxes and Costs Challenge the European Market.** *Tobacco Reporter* 111(7): 32-34, 36, July 1984.

Basic changes in consumer demand, in addition to higher taxes and increasing manufacturing costs, have forced European cigarette producers into marketing blended cigarettes (which most smokers now favor). In France, where taxes currently account for 77 percent of the retail price of the most popular brand, imports (of blended cigarettes) continued to gain in 1983. In the Netherlands, cigarettes for export make up almost half of total production, and there is concern that the 25 percent increase in the French import tax will endanger its biggest market. In addition, domestic consumption declined 6.5 percent in 1983. Cigarette production in Austria reached its highest level, but consumption of domestic brands is declining in favor of foreign brands (many produced locally under license). While exports declined in most traditional markets, Austria is actively pursuing cooperation with Japan in developing new brands. The price difference between imported and domestic brands in Italy is the main reason for the small shift in market shares in 1983. Most imports are American brands manufactured in Germany or the Netherlands. Uneven price increases (tax-related) in Greece have made imported cigarettes even more expensive compared with domestic brands, virtually assuring the market for domestic brands. Swiss domestic consumption is relatively stable. The 2 percent market growth is mainly from increased tourist and border sales. Despite a small decline in 1983, sales in Belgium remained higher than the average volume registered for 1976 through 1980. As a result of an excise tax increase in Denmark, domestic consumption declined 7 percent and exports 12 percent.

84–1937. Umur, H. **Turkish Tobacco Production Jumps 8.5 Percent, But Quality Mixed.** *Tobacco International* 186(13): 45-46, June 8, 1984.

Rainy weather is blamed for the poor quality of the 1983 crop in Turkey, but high prices for top quality leaf could mean a better crop this year. Stocks of old crops held by the State monopoly have declined, and in the first 3 months of 1984, exports were up sharply over the same period in 1983. Although cigarettes can now be imported, duties and taxes will make them approximately three times as expensive as top domestic brands. The first imported brands arrived in early April; it remains to be seen how well they will sell.

LEGISLATION

84–1938. Chapman, S.; McKay, B.; Kirby, R. **Good for the Goose—Good for the Gander?** *Media Information Australia* 31: 47-54, February 1984.

The advertising self-regulatory system in Australia recently dealt with two complaints concerning cigarette smoking. The tobacco industry lodged a complaint about two advertisements from a large-scale smoking control program, resulting in one being effectively banned. Complaints made by a consumer group about three cigarette advertisements were not upheld. Details of the complaints and their outcomes are given. It is concluded that considerably greater latitude in what was judged to be acceptable was afforded cigarette advertisers than the smoking control program. The chairman of the Advertising Standards Council (Kirby) responds to the critique, justifying the decisions of the council, the choice of council members, and refuting the claim that greater latitude was given to cigarette advertisers. The original authors review the response and discuss three problems seen as central to the dispute: the composition of the Council; its closed procedures and determinations; and anomalies in the standard of evidence it has accepted. (Auth. Abs. Mod.)

84–1939. Hersh, S. P. **Cigarette Smoking Injuries: A Theory for Recovery Against the Federal Government.** *Trial* 19(4): 72-75, 99-100, April 1983.

The tobacco industry has thus far successfully defeated every attempt to impose civil liability on it for smoking-related cancers that are responsible for approximately 100,000 deaths annually. Ironically, the required notice on all cigarette packages is currently the major obstacle to a finding of implied warranty of merchantability in the industry. A possible legal case against the Government under the Federal Tort Claims Act (FTCA), whereby Federal Trade Commission (FTC) employees would be liable on the grounds that they failed to perform adequately and with due care in preventing false advertising, is outlined and difficulties in presenting such a case are reviewed. Although congressional policy is to adequately inform the public of smoking's hazards (and false advertising violates this policy), the FTC and its employees do not have the option to accept or reject the policy; they only carry it out, and lack of action to ameliorate the problem is not an abuse of the discretionary function exception of the FTCA.

84–1940. Ireland, K. T. **Cigarette Smoking Injuries: A Theory for Recovery Against the Federal Government.** *Trial* 19(4): 67-72, 97-99, April 1983.

The imposition of liability on the Federal Government for its contribution to the cigarette-cancer dilemma is neither a promising legal possibility nor a desirable socioeconomic objective. In the case of smokers who have developed cancer, the discretionary function and misrepresentation exceptions of the Federal Tort Claims Act would protect the government from liability in a suit claiming negligent failure to warn. For cancer victims exposed to cigarette smoke in the workplace, chances of recovery are greater, but plaintiffs would have to prove that cigarette smoke actually caused cancer. Should courts allow recovery against the Federal Government under any theory, the taxpayer would shoulder an even greater financial burden than now. Further, if smokers could rely on Government compensation if they developed cancer, they would have no incentive to reduce tobacco consumption. Finally, allowing recovery might cause the Government to repeal its cigarette labeling and advertising statute or to withdraw from the area of industrial regulation, which might lead to an increase in smoking. Internalization, a better solution, would place the cost of cigarette cancer where it belongs, with the tobacco industry and with the consumers of its products. (Auth. Abs. Mod.)

84–1941. U.S. Senate, 98th Congress, 2nd Session. **A Bill to Establish an Interagency Committee and a Technical Study Group on Cigarette Safety.** Introduced October 6, 1983, 98th Congress, Second Session, by Senators Heinz, Danforth, Cranston, Boschwitz, Dodd, Stafford, Proxmire, Inouye, Hawkins, Roth, Cohen, and Sasser, Revised May 25, 1984, Senate No. 1935, 1984, 16 pp.

The revised bill would establish an Interagency Committee on Cigarette and Little Cigar Fire Safety that would consist of (1) the Administrator of the United States Fire Administration

(Chairman), (2) the Assistant Secretary of Health of the Department of Health and Human Services, and (3) the Chairman of the Consumer Product Safety Commission. The committee would direct, oversee, and review the work of the Technical Study Group that would consist of 15 scientific and technical representatives of various medical, fire protection, manufacturing, research, and tobacco-related organizations. The group would attempt to determine the technical and commercial feasibility, economic impact, and other consequences of developing cigarettes and little cigars that would have a minimum propensity to ignite upholstered furniture and mattresses. A status report would be submitted a year later to the Senate and House of Representatives and a final report would be prepared within 30 months.

GENERAL

84–1942. Blyth, M. **Cigarette Ads in the *Ladies Home Journal*. (Letter).** *New England Journal of Medicine* 311(1): 52, July 5, 1984.

In response to a concurrently published letter (*New England Journal of Medicine* 311(1): 51-52, July 5, 1984) that criticized the *Ladies Home Journal* for not emphasizing the health hazards of smoking, it is pointed out that in three of the last six issues, major articles warned women of the risks of smoking: (1) an item in the January 1984 issue stated that smoking accounted for 25 percent of cancer deaths in women; (2) "Winning the War Against Cancer" (May 1984) estimated that lung cancer could be reduced 90 percent by avoiding tobacco; and (3) "What's Wrong When the Doctor Says Nothing's Wrong" (June 1984) advised women to eliminate toxins, noting that cigarettes are among the most lethal toxins legally available. The magazine's editor-in-chief also believes that cigarette companies have the right to advertise, the magazine has the right to accept those advertisements, and the implication that the increasing number of pages devoted to cigarette ads is related to the increasing rates of lung cancer incidence in women is of questionable logic. See also 84-1950.

84–1943. Chandler, W. U. **Improving World Health: A Least Cost Strategy.** Worldwatch Paper No. 59, Washington, D.C., Worldwatch Institute, July 1984, 68 pp.

Least-cost health strategies will accord priority to preventive and primary methods designed to attack the world's leading causes of unnecessary death. Tobacco control will require many decisions on many governmental levels. Strong health warnings and the prohibition of advertising are critical first steps. A tax on cigarettes of 2 dollars/pack would discourage smoking and place costs on smokers. Restricting public smoking would protect the health of nonsmokers. Progress will be slow and leadership at the highest levels of government will be critical. Parties with vested interests (the medical insurance industry for pecuniary reasons and the health care industry for ethical reasons) can take the lead in pressuring governments to implement public health priorities. Citizens and health promotion organizations will need broad support, both financial and political, to be effective. Childhood infections can be sharply cut by extending primary health care to the world's poor women and children. The cost would total only 10 billion dollars/year, less than two-tenths of a percent of the world's annual economic output. Where the incidence of diarrheal, tropical, and parasitic diseases are highest, tens of billions of dollars worth of investments in wells and toilets will be necessary, but cost effective. The toll of heart disease and cancer in middle age can probably be halved with diet modification and control of smoking. Educational campaigns for reducing fat and cholesterol consumption, coupled with taxes on tobacco and restrictions on public smoking, can help extend millions of lives into old age. But the best hope of low-cost cures for high-cost diseases lies in basic science. Fortunately, the additional cost of research and development would be comparatively low. (Auth. Abs. Mod.)

84–1944. Chwat, J. **Tobacco in Washington. An Interview With E. de la Garza, of the House Agriculture Committee.** *Tobacco International* 186(13): 52, 54, 56, June 8, 1984.

The chairman of the House Agriculture Committee states that tobacco legislation will be a continuous battle and that removing tobacco from the farm program is more than a health issue. However, he does not expect any major legislation to be considered during this session. The Congressman thinks U.S. products are competitive in terms of supply and quality, but not always in price, and is actively attempting to open up cigarette and leaf markets for U.S. tobacco in Japan, France, and other nations. He also does not believe tobacco companies are being forced to diversify; diversifying is an economic necessity for all industries. He is aware of cigarette smuggling activities at the Mexican border, but feels the numbers involved are not of major impact. Smuggling is a complicated issue, and customs regulations have been tightened. The Congressman also supported the Caribbean Basin Initiative bill (foreign import legislation, including tobacco), although he was not satisfied with all items in the law.

84–1945. Collishaw, N. E.; Myers, G. **Dollar Estimates of the Consequences of Tobacco Use in Canada, 1979.** *Canadian Journal of Public Health* 75(3): 192-199, May-June 1984.

For 1979, economic losses attributable to tobacco use in Canada are estimated at 5.2 billion Canadian dollars, over 2.0 billion more than consumer expenditures for tobacco products during the same year. Estimated losses include lost income owing to smoking-attributable mortality, 3.3 billion; smoking-related disability, 616 million; hospitalization, 1.1 billion; physician services, 22 million; and damages from cigarette-related fires (not including the cost of fighting the fires), 84.9 million. Estimates are judged to be conservative because factors such as the cost of passive smoking, building ventilation and maintenance, fire insurance, and pharmaceutical products were not included. In 1979, Canadians spent 3 billion dollars on tobacco products, with 29 percent going to the Federal Government and 22 percent to provincial governments in taxes. It is noted that 17 percent of all adult deaths and 5 percent of all adult disability days are attributable to tobacco, constituting a strong argument for antismoking actions.

84–1946. Cumper, G. E. **Not So Simple. (Letter).** *Nature* 308(5958): 397, March 29, 1984.

A previous article (*Nature* 306(5942): 418, December 1, 1983) that presented a graph of tobacco consumption vs. advertising in Norway to support a suggested ban on promotion is criticized. It is noted that, contrary to the assumption that an advertising ban would reduce consumption, the charted decline began before Norway's prohibition of advertising. Also, the implication that the increase in consumption between 1950 and 1970 was caused by advertising is questioned. An examination shows that the ratio between per capita consumption and per capita incomes had been declining since 1950. Such economic factors should be considered in policy decisions because the antismoking effort will only be weakened by incomplete statistics. See also 84-1298.

84–1947. Cunningham, J. I. **Reviving Medicare: A Suggestion. (Letter).** *Canadian Medical Association Journal* 130(7): 846-847, April 1, 1984.

As a solution to the monetary problems of the Medicare system, it is proposed that expenses incurred by health care costs be borne by the three major causes: smoking, alcohol consumption, and motor vehicle accidents. Approximately 30,000 deaths in Canada each year are attributable to smoking. The costs of postponing these deaths and of the treatment of cigarette-related burn victims justify levying a health tax on tobacco products. It is also suggested that a label stating that "A health tax of — percent has been levied to pay for the treatment of illnesses attributed to the use of this product" may have more preventive impact than the health warnings currently in use.

84–1948. Frogatt, P. **Tobacco Facts. (Letter).** *New Scientist* (1403): 50, March 29, 1984.

An earlier report (*New Scientist* 101(1401): 4, March 15, 1984) is criticized for inaccuracies concerning the relationship between the tobacco industry and the Independent Scientific Committee on Smoking and Health. The committee formulates advice on scientific data and no attempt to influence its decisions would be countenanced. Disbursement of funds, including those from the Tobacco Advisory Council, is at the discretion of the trustees, and grants are made without reference to the tobacco industry or to the Government. The committee relies on its independence and perceived integrity for its effectiveness. See also 84-1949.

84–1949. Harriman, E. **Tobacco Firms Vet Smoking Studies.** *New Scientist* 101(1401): 4, March 15, 1984.

Only 10 percent of monies provided by the tobacco industry for research into smoking and health has been spent, because of industry objections to research proposals. The industry's donation is part of a voluntary agreement with the British Government, whereby the Government promises not to introduce legislation curbing tobacco advertising in exchange for funding of independent research into the health effects of tobacco products. The money is being held by the Tobacco Products Research Trust. A total of 300,000 pounds, of a 3 million pound program, has been allocated. All of the funds have gone to members of the Independent Scientific Committee of Smoking and Health, which is responsible for deciding how the donation is to be spent. The committee has stated that there is no conflict of interest in awarding funds to committee members, as there are relatively few groups equipped to do the needed research, and reconciling health interests with the terms set by the tobacco industry is proving difficult.

84–1950. Kitchens, C. S. **Cigarette Ads in the *Ladies' Home Journal*. (Letter).** *New England Journal of Medicine* 311(1): 51-52, July 5, 1984.

The *Ladies Home Journal* is criticized for warning about the dangers of chocolate, coffee, and alcohol, but not mentioning cigarettes, in its 100th-anniversary issue. The issue featured women's health as "the story of the century" and correctly stated that the *Journal* has helped inform the public about the importance of breast self-examination, pap smears, and contraception. Attention was also given to nutrition, osteoporosis, microwave cooking, and exercise. However, lung cancer, the only epidemic involving this audience, is not mentioned, although a small paragraph notes that lung cancer will overtake breast cancer as the primary cause of cancer mortality among women. The number of pages devoted to cigarette advertising rose from 0.1 percent in 1960 (2 pages out of 2,022) to 8.8 percent in 1980 (180 pages out of 2,044). During this time, lung cancer in women increased from 6/100,000 to 22/100,000. Physicians are urged to remove from waiting rooms any magazines that carry cigarette advertisements.

84–1951. Lutschg, J. H. **Smoking and Public Policy. (Letter).** *New England Journal of Medicine* 311(4): 260, July 26, 1984.

In addition to the tax revenues generated by cigarettes and the "politics of regionalism" suggested as being responsible for the Government's long-term benevolent posture toward smoking (*New England Journal of Medicine* 310(8): 539-544, February 23, 1984), it is pointed out that lobbying efforts and campaign contributions by the Tobacco Institute and the Tobacco People's Public Affairs Committee play an important part. No action has been taken on the Comprehensive Smoking Prevention Education Act of 1982, at least in part because of concern that passage of the bill might heighten tobacco companies' liability for their products. Rather than "a benign government policy," it appears more reasonable to view such actions as a malignant policy toward the American public's health. See also 84-1302.

84–1952. Robbins, A. **Creating a Progressive Health Agenda. 1983 Presidential Address.** *American Journal of Public Health* 74(8): 775-779, August 1984.

In an address about health issues for the 1980s, it is noted that the health of the health industry has become a political issue, with emphasis on cost containment rather than on health. However, the following new agenda is suggested that would contribute efficiently to a healthy U.S. population: (1) disease eradication that would encompass all efforts to improve the health of the American people (control of smoking would prevent many diseases, but prevention and cessation programs have been grossly underfunded and the Government has never used the means at hand to reduce the profits associated with cigarette sales); (2) primary care, which is the key to making medical services effective; (3) long-term care financing, needed because of the rapidly growing elderly population; and (4) occupational and environmental health, where the productive part of the economy interacts with the health of the people. Unfortunately, cost containment and cutbacks will be the constant refrain in all national political debates, but cost containment is not a health issue. Good health policies will restrain both disease and costs and will promote public health.

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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Greiber, R., 84–0561
Greig, C. C., 84–0635
Greitemeyer, M., 84–0736
Grell, G.A.C., 84–1343
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Gupta, M. C., 84–0688
Gupta, R. S., 84–0813
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Gusella, J. L., 84–1475
Gust, S. W., 84–0562, 84–0583
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Gutenmann, W. H., 84–1639
Guthenberg, C., 84–0053
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Haber, W., 84–1281
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Hackney, J. D., 84–0476
Haddow, J. E., 84–1367
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Haenszel, W., 84–0434
Hag, F.-L., 84–1889
Hagenmuller, F., 84–0851
Hagiwara, T., 84–0154
Haglind, P., 84–1774
Hahn, S., 84–1670
Haigh, N. Z., 84–0500
Haile, B. M., 84–1843
Haines, A. P., 84–0724
Hakulinen, T., 84–0446
Halawa, B., 84–1452
Haley, N. J., 84–0037, 84–0296, 84–0563, 84–1035
Halikas, J. A., 84–1872
Hall, J. B., 84–0939, 84–0940, 84–0941, 84–1268, 84–1271, 84–1272, 84–1912
Hall, R., 84–1292, 84–1917
Hall, S. M., 84–0019, 84–0683, 84–1663, 84–1861
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Hallgrimsson, J., 84–0098, 84–0773
Hallmark, R. K., 84–0042
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Hamermesh, D. S., 84–0238
Hamermesh, F. W., 84–0238
Hamilton-Stewart, P. A., 84–0742
Hammad, Y. Y., 84–0163
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Hammond, S. K., 84–1129
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Hand, W. L., 84–1019
Handy, R. W., 84–0383
Hanisch, R., 84–0777
Hankin, J. H., 84–1066
Hanks, D. T., 84–1873
Hannan, M. A., 84–0406
Hanninen, O., 84–0709
Hansen, J. C., 84–0032
Hansen, P. L., 84–1543
Hansen, S. H., 84–0378
Hansen, W. B., 84–0565
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Harada, T., 84–1020
Hardenberg, J. P., 84–0515
Hardy, R. J., 84–0764
Harken, D. E., 84–0181
Harley, N. H., 84–1386
Harman, A., 84–1918
Harman, M., 84–1184
Harman, M. L., 84–0237
Harper, P. G., 84–1711
Harper, P. H., 84–1286
Harriman, E., 84–0008, 84–0339, 84–1949
Harris, B. S., 84–0383
Harris, C. C., 84–0099, 84–1733
Harris, J., 84–0913
Harris, J. E., 84–0239
Harris, M., 84–0240
Harris, R., 84–1707
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†Hauni-Werke Korber and Co. KG, 84–1265, 84–1283, 84–1284
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Hawkins, C. M., 84–0764
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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Hayashi, Y., 84–0760
Hayden, J., 84–1756
Hayden, S. P., 84–1756
Hayes, E. B., 84–0946
Hayes, T. M., 84–0828
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Hazon, J., 84–1105
Health and Welfare Canada, 84–0276
Hearne, J. T., 84–0907
Heath, C. W., Jr., 84–1665
Hebel, J. R., 84–0845, 84–1681
Hecht, S. S., 84–0373, 84–0677, 84–0678, 84–0682, 84–0693, 84–0756, 84–1337, 84–1703
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Hedrick, B. M., 84–0608
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Heeren, T., 84–0808
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Henningfield, J. E., 84–0035, 84–0568, 84–1211, 84–1212, 84–1510, 84–1515, 84–1516
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Herd, J. A., 84–0820
Heribanova, A., 84–0068
Hering, R., 84–1814
Herity, B., 84–0101
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Herman, S., 84–0484
Herning, R. I., 84–0019, 84–0683, 84–0702, 84–1663
Herraiz, M., 84–1155
Herrmann, H., 84–1108
Herrmann, U., 84–0198
Hersey, P., 84–1022
Hersh, S. P., 84–1939
Herve, M., 84–0536
Hetzel, D. J., 84–1183
Heuch, I., 84–0758
Hibbitts, C. H., 84–1282
Hibble, A. G., 84–0364
Hickey, N., 84–0171, 84–0518, 84–0805, 84–1454
Hickey, R. J., 84–0438, 84–1501
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Hietanen, E., 84–0847
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Higgins, C. E., 84–0679
Higgins, I.T.T., 84–0147, 84–0156, 84–0737, 84–1065
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Hildebrandt, J., 84–1127
Hilgenfeldt, J., 84–0036
Hill, C., 84–1054
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Hill, P., 84–0037
Hillbom, M., 84–1799
Hiller, F. C., 84–0009
Hillerdahl, G., 84–1414
Hillerdal, G., 84–0439
Hillhouse, A. J., 84–0273
Hillis, L. D., 84–0816
Hindi-Alexander, M., 84–0926
Hinds, M. W., 84–1066
Hingson, R., 84–1474
Hinzmann, A., 84–1283
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Hjermann, I., 84–0508
Hobbs, M.S.T., 84–0510
Hoch-Ligeti, C., 84–1387
Hochhauser, M., 84–0579
Hodge, R. L., 84–1458
Hodges, D., 84–1539
Hoefel, O. S., 84–0038
Hoey, J. R., 84–1716
Hofer, V., 84–0180
Hoffman, D., 84–1639
Hoffman, I., 84–0001
Hoffmann, C. O., 84–0093
Hoffmann, D., 84–0127, 84–0296, 84–0356, 84–0386, 84–0670, 84–0672, 84–0673, 84–0677, 84–0678, 84–0680, 84–0681, 84–0682, 84– 84–0756, 84–0998, 84–1535, 84–1635
Hoffmann, R. G., 84–1872
Hogan, T. J., 84–0786, 84–1406
Hogetveit, A. C., 84–1180
Hogg, J. C., 84–0144, 84–0490
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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Hsu, F. S., 84–0363
Huba, G. J., 84–0229
Huberman, E., 84–1668
Hudgens, R. W., 84–0063
Hudson, L. D., 84–1758
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Hugh-Jones, P., 84–1110
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Hughes, J. A., 84–1110
Hughes, J. M., 84–0163
Hughes, J. R., 84–1904
Hukkelhoven, M.W.A.C., 84–1667
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†Imperial Group Ltd., 84–1264
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Institute of Paper Chemistry, 84–0297
†International Flavors and Fragrances Inc., 84–0631, 84–0927, 84–0928, 84–0929, 84–0930, 84–0931, 84–0934, 84–0935, 84–0936, 84–0938, 84–0939, 84–0940, 84–0941, 84–0942, 84–1254, 84–1255, 84–1256, 84–1257, 84–1258, 84–1259, 84–1260, 84–1261, 84–1262, 84–1263, 84–1268, 84–1169, 84–1271, 84–1272, 84–1559, 84–1560, 84–1911, 84–1912
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Ippoliti, A., 84–0217
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Ireland, K. T., 84–1940
Isager-Sally, L., 84–0205
Ishiguro, S., 84–1641
Ishikawa, T., 84–0703
Ishiyama, I., 84–0039
Ishizaki, T., 84–0401
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Jacobson, B., 84–0341
Jacobson, E. D., 84–1794
Jacqueson, A., 84–1140
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Jaffe, J. H., 84–0040, 84–0055
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Jagenburg, R., 84–0046
Jain, S. K., 84–0145
Jamborcic, V., 84–0895
James, J., 84–0206
James, L. S., 84–1165
Janoff, A., 84–0146, 84–1114
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Jenkins, B., 84–1801
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Johnson, L. F., 84–1186
Johnson, L. P., 84–0449
Johnson, M.R.D., 84–1213
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Johnson, P. R., 84–1588
Johnson, R. E., 84–0035
Johnson, W., 84–0073
Johnson, W. D., 84–0091
Johnston, A., 84–0867
Johnston, A. W., 84–0542
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Jones, C. A., 84–1668
Jones, J. G., 84–1767
Jones, J. R., 84–0147

***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Jones, K. G., 84–0408
Jones, L. A., 84–0214
Jones, N. L., 84–0187
Jones, R. N., 84–0163
Jones, P. T., 84–0019, 84–0370, 84–0683, 84–0702, 84–1663
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Joossens, J. V., 84–1363
Jooste, P. L., 84–0824
Jordaan, P.C.J., 84–0824
Joseph, H. D., 84–1284
Joseph, L. B., 84–0411
Joseph, M., 84–0801
Journal of Traffic Medicine, 84–0219
Joyce, H., 84–1780
Jozala, E., 84–1067
Jugdutt, B. I., 84–0502
Juhl, R., 84–1728
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Kabat, G. C., 84–1068
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†Kabushiki Kaisha Advance Kaihatsu Kenkyujo, 84–0640
Kadar, D., 84–0065
Kaelin, A. C., 84–0708, 84–1349, 84–1476, 84–1817
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Kagawa, Y., 84–1664
Kagen, S. L., 84–0044
Kageyama, H., 84–1685
Kahan, J. P., 84–1892
Kahn, P. L., 84–0979
Kaij, L., 84–0841
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Kalliokoski, P., 84–0709
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Kashani, J. H., 84–0183
Kashani, S. R., 84–0183
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Kato, S., 84–0269
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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Reed, D. M., 84–1467, 84–1470
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Rees, T. D., 84–1851
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Rodriguez, I., 84–1499
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Ronnqvist, P., 84–1151
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Russell, J., 84–0580
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Sachero, A., 84–0498
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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Sadoul, P., 84–0445
Sadur, C. N., 84–1836
Saengchan, S., 84–1359
Saetta, M., 84–1747
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Schenker, M. B., 84–0794, 84–0795
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***Individual has contributed to this reference but is not listed as part of the main citation.**
†Patent assignee.

Selkirk, J. K., 84–0721, 84–1668
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Selli, C., 84–0762
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Skellern, G. G., 84–1174
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Solomon, R. J., 84–1850
Solomons, N. W., 84–0793
Somerville, K. W., 84–1493
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*Individual has contributed to this reference but is not listed as part of the main citation.
†Patent assignee.

Sonnenberg, A., 84–1194
Sonnenfeld, G., 84–0063
Sorensen, B. L., 84–1074
Sorsa, M., 84–0034, 84–0409, 84–1038, 84–1362
Sos, T. A., 84–0512, 84–1148
Sotelo, J. M., 84–1391
Soto, E., 84–0089
Souhami, R. L., 84–1711
Soutar, C. A., 84–0798
South African Medical Journal, 84–1245
Sparros, L., 84–0122
Sparrow, D., 84–0521
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Spealman, R. D., 84–0593, 84–1510
Speir, W. A., Jr., 84–1742
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Stanwell-Smith, R., 84–0724
Stapleton, J., 84–1252, 84–1555
Staszewska-Barczak, J., 84–1806
Statistical Bulletin, 84–0905
Stavenow, L., 84–1364
Stead, W. W., 84–0485
Stedronska, J., 84–0724
Steele, I., 84–1603
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Steenhoek, A., 84–0725
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☆ U.S. GOVERNMENT PRINTING OFFICE : 1985 O - 474-271